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THE SURGICAL ENTITY OF MUSCLE RECESSION *

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BROOKLYN

In the ARCHIVES OF OPHTHALMOLOGY for 1922 [1] and in the *Transactions of the Ophthalmological Society of the United Kingdom* for 1925,[2] I outlined the principles of what I designated as the operation of recession. It was devised because of the belief that tenotomy, which in contrast to advancement had never been an operation of precision, could be made so and its usefulness greatly augmented and safeguarded. It was also born of the conviction derived from experience with the combined operation that a large proportion of the corrections attributed to advancement, and especially to resection, were in reality brought about by the tenotomy, and if the tenotomy could be extended under safe conditions, computed and graded, and the end of the muscle sclerally fixed as in advancement, the yield in corrections would be greatly increased and stabilized.

It was evident at the beginning that if a useful and reliable investigation was to be undertaken which would prove or disprove the foregoing assertion, it would have to be carried out as a single entity, separate from any combined procedure, and on this basis the work was done. This entity I named "recession" as the antithesis of advancement, and when mentioning the term "recession" I shall use it as the embodiment of the entity, the principles and technic of which are outlined and described in these pages.

One has only to glance at the history of tenotomy since its inception to be cognizant of the surgeon's manifest desire to use it, and to be aware of his recognition of its corrective value and also, as evidenced by the multiplicity of devices (curb, guarded, cross-cut, buttonhole, etc.), of his endeavor to stabilize it. That these efforts have been unsuccessful is revealed by the fact that tenotomy has been virtually abandoned as a primary procedure.

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* Submitted for publication, March 30, 1931.

* Read before the Section of Ophthalmology, New York Academy of Medicine, March 16, 1931.

1. Jameson, P. C.: Correction of Squint by Muscle Recession with Scleral Suturing, Arch. Ophth. **51**:421 (Sept.) 1922.

2. Jameson, P. C.: The Principles of Muscle Recession with Scleral Suturing as Applied in the Correction of Squint, Tr. Ophth. Soc. U. Kingdom **45**:405, 1925.

Three things that I believe the experience of my work has saliently demonstrated are: 1. Recession has corroborated both the corrective value of tenotomy and its evident shortcomings. 2. With the bulwarks of safety which it has introduced and utilized, the entity of recession replaces tenotomy by a procedure of great primary value and high corrective return. 3. Recession is the operation of selection in cases in which tension and hypertonicity are the predominating features.

I shall outline the subject under three general headings—the problems which presented themselves for solution, the procedure adopted and the explanation of the steps of that procedure in detail—and shall then add some brief comments on recession in connection with advancement, postoperative treatment and end-results, and some final conclusions.

A FEW OF THE PROBLEMS

In groping for the truths offered by the premise of a single entity, and in bringing about the actualization of such truths, so radical in their intent, it was apparent that many difficulties would be encountered. This field was unexplored.

1. I would have to overcome the traditional stumbling block that the sclera could not be safely sutured at the equator on account of the thinness of this region.

It would be necessary to establish the fact that structurally this region was suitable for scleral suturing, and to make a practical demonstration of the fact that the venous system was not a deterrent, as no evidence showing the extent or limits of such safety had been made. Also could a technic be devised for making scleral suturing safe if the sclera proved to be good suture material? This would have to be established. On determining the principle of this safe technic, what instrumentation and needles for scleral splitting which admitted introduction of suture and needle, not alone into this superficial covering but well into the fibrous structure, could be originated?

2. What would be unnecessary mutilation with regard to muscle and flap?

In this connection should the muscle be isolated and stripped of its fiber or should it be receded in the flap with little disturbance of its normal surroundings? Again the question as to whether and how tenotomy could be more freely extended with scleral fixation had to be answered. What was the most suitable manner of accomplishing this? Was there a better way than the usual incision into the capsule and muscle as in tenotomy? How could convergence best be protected

and caruncular retraction be prevented and at the same time the muscle be permitted to assume its flattened condition against the globe?

3. What computations of gradation and measurements were to be used?

No definite ideas of how far one could recede had ever been worked out. One would be on dangerous ground in the region of the venae vorticosae. How was the amount of recession to be definitely established? The definite equatorial position in a given case, and its limitation as a landmark back of which a recession could not be done, had to be ascertained. What correction was obtainable by a single or bilateral recession? Also on pursuing the subject, striking anatomic conditions of muscle and capsule confronted one. How best could this knowledge be utilized in the computation of gradation and correction?

These and other details which seem plain now were clouded in doubt and uncertainty.

THE PROCEDURE ADOPTED

The principles of this entity or system of recession are distinct and revolutionary. The reader will bear in mind the purpose, which is to take tenotomy out of the field of unreliability and place it on a safer and sounder basis, making it an operation of precision and greatly extending its usefulness as well as enlarging its field of correction. The means followed to obtain this were: (1) definite scleral fixation of the end of the muscle by a safe technic of scleral suturing; (2) extended tenotomy under secure conditions; (3) the practice of accurate gradation after careful computation; (4) a technic that disturbs the anatomic relationship in the least degree by retroplantation in flap and nonmutilation of tissues above and behind the caruncle, contributing to the protection of convergence and prevention of caruncular retraction; (5) a study of the exposed muscle and capsule and the obtainment of knowledge whereby obstructive conditions contributing to the deformity could be remedied.

The following steps of the operation embodying these principles were evolved after many trials and experiments, and I feel now are the best adapted to smooth technic.

A marking on either side of the limbus in the horizontal axis when the eye is in the primary position will make for a better understanding of the position of the axis of the muscles should torsion occur under anesthesia. It will enable the operator to obviate irregular incisions or buttonholing of the muscle, an accident that occasionally happens. A dot from a toothpick immersed in a concentrated solution of gentian violet (10 per cent in equal parts of alcohol and water) is suitable

for the purpose. The conjunctiva should be dried with a cotton pledget when this solution is used, otherwise it will run. The solution is also excellent to mark the sclera where the entrance of sutures is to be defined.

Local anesthesia is desirable for operation. The operator may use the anesthetic that he prefers and in the manner that his experience indicates. Instillation may be supplemented by subconjunctival injections. I use 2 per cent procaine hydrochloride for this. Although it takes longer to effect anesthesia, it is efficient. These injections are best made above and below if the lateral muscles are operated on, care being taken that the bleb does not invade the field of operation. Slight massage through the lid will flatten the bulge. A small pledget soaked in 4 per cent cocaine and placed on the field between the lid and bulb for a few minutes before operation will give much more anesthesia than repeated instillations during operation.

The conjunctival incision is vertical and is about 7 or 8 mm. in length following the curve of the semilunar fold, and not in the usual place over the insertion of the muscle.

The field of operation is now exposed, the flap being dissected and turned over toward the cornea. The conjunctiva over the caruncle need not and should not be disturbed for reasons outlined later.

The capsule is now buttonholed (with blunt-pointed Stevens' scissors, or with sharp scissors if the operator is adept) by pinching up a fold of capsule with tooth forceps. The point for buttonholing should be well behind the insertion of the muscle (about 5 mm.) and about 3 mm. above the muscle in the region of the equator, this for reasons explained later. The forceps should not let go the pinch of capsule until the strabismus hook has been inserted in the buttonhole and has found its way under the muscle. If this is not observed, the hole may be lost while the operator is looking for the strabismus hook. The scissors may precede the hook through the buttonhole, preparing a way for it and separating the trabeculae from the globe.

The muscle and capsular flap are now outlined. The muscle being undermined, the hook is swept under it well beyond the opposing border of the muscle, where it is again buttonholed at a point corresponding in distance from the muscle to the point primarily buttonholed. The muscle is now thoroughly undermined, and capsular incisions markedly diverging are regulated according to the amount of recession sometimes required behind the equator. These incisions to complete the flap are also carried from the point of entrance and exit of the strabismus hook to meet the muscle at the outlines of its insertion. This will outline the flap to be receded. The female blade of muscle forceps is now introduced beneath the muscle (from below in the right eye

and from above in the left), if the interni are being receded, and the muscle is clamped and severed, care being taken not to snip the globe at this point under insertion where the sclera is of paper thinness.

The sclera is now thoroughly exposed, the muscle separated from the sclera to the base of the flap, and the débris and blood are sponged away. The sclera then appears white and glistening, and presents an unobstructed field for the introduction of the scleral sutures. Small venous extravasations may be controlled by an epinephrine pad. Rarely does one have the anomaly of venae vorticosae emanating from the region of the equator. They are always visible and easily avoided in introduction of the needles.

The measurements and gradation are now determined. The distance from the insertion of the muscle (presently to be described) that the operator desires to recede the muscle is measured in millimeters, and the point is noted on the sclera.

Three single-armed sutures are provided. The first, the central one, is introduced from without the conjunctival lip nearest the caruncle; it then perforates the outer surface of the muscle end 1.5 mm. below the center and 1.5 mm. back of the end of the tendon. The needle then is passed through the muscle from the under surface, 1.5 mm. above the center of the muscle, and brought out on the upper surface. The thread is looped around and behind the suture where it first perforated. This central suture is now made to split the sclera, taking a 2 mm. bite, and being carried forward it perforates, on its under surface, the conjunctival flap adjacent to the cornea. The two remaining wing sutures pass in transit from the outer surface of the caruncular conjunctiva, muscle end, sclera and conjunctival lip nearest the cornea. Their perforations through the caruncular conjunctiva, sclera, end of the muscle and conjunctival lip adjacent to the cornea should be made sufficiently well apart to insure a broad attachment of the muscle to the globe and permit the closure of the extended primary conjunctival incision. They should be at least 3.5 mm. apart. The sutures are now carefully separated, the toilet of operation completed and the muscle approximated to its new scleral attachment. The tying of the sutures firmly holds the muscle to its attachment and at the same time closes the conjunctival opening.

Perhaps the best way to give the surgeon a basis of understanding of this procedure is to take up these steps in detail, embodying at the same time some of the aforementioned difficulties and problems encountered and overcome, with the reasons for the adoption of the technic just described.

THE CONJUNCTIVAL INCISION

The incision should be vertical. A horizontal incision is not suited for recession in the sense in which it is used in these pages. The operator will find himself cramped for room if it is used; exposure will be imperfect and broad attachment difficult. The incision made in the usual position, that is, over the end of the muscle, is not suitable; it should be made at the point to which the end of the muscle is to be receded, that is, slightly in front of the semilunar fold; otherwise the lip of the corneal side of the conjunctiva will not stretch to meet the caruncular lip and will tear out, leaving the sclera exposed. This is a common early mistake (fig. 1).

The conjunctiva should be undermined to the corneal margin. The caruncular conjunctiva with its succulent underlying tissue will always

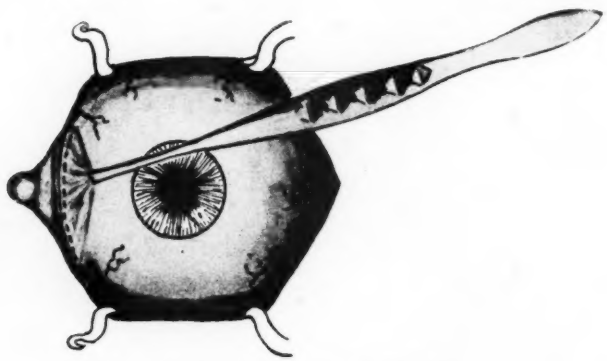

Fig. 1.—Conjunctival incision.

stretch without difficulty and should not be separated from the underlying tissue. The lips of the corneal wound do not unite by primary union, and properly so, as any attempt to coapt them too closely will dam back the serosanguineous discharge, always copious from surgical disturbance of muscle and capsule, which should freely drain. The through-and-through suture arrangement which closes the wound at the same time that the muscle and scleral suture is tied is sufficient if the operation has been technically sound. If a slight gap should remain, supplementary conjunctival sutures may be used. Not infrequently during the healing process a white line of sclera is seen at the bottom of the sulcus between the turgid lips of the conjunctiva. This is of no significance as the epithelium proliferates over as soon as the reaction has subsided. The dissection and laying over of the conjunctival flap toward the cornea serve another valuable purpose, that of complete exposure of the operative field. It is here that one is able to examine the capsule and, by traction in different directions, ascertain

its tension or laxity. It also permits one to buttonhole the capsule near the equator, a much easier position than nearer the insertion of the muscle where it coapts closer to the globe and not much knowledge can be obtained of its physical condition (fig. 2).

Little attention has been devoted in the past to the exposed field of operation. Computations as to what shall be done are largely based on objective symptoms and tests prior to operation. The dissection of the conjunctival flap and the thorough exposure that this produces enable one to make a study of the actual muscular and capsular con-

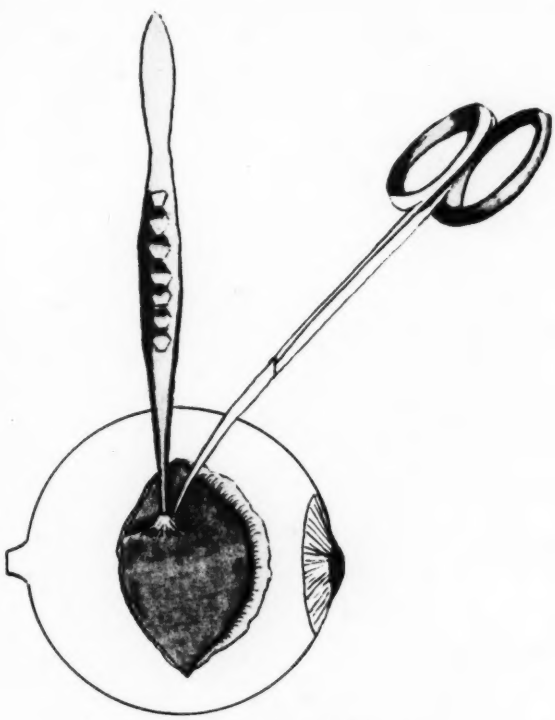

Fig. 2.—Buttonholing capsule.

ditions that exist at the time of operation. This contributes in no small degree to the knowledge of how and to what extent the surgical procedure will be conducted.

CAPSULAR AND FASCIAL CONDITIONS

After exposure, if the capsule can be grasped at a point behind the equator clear of the muscle its laxity or tautness can be ascertained. Some capsules can be dragged 5 or 6 mm. in any direction when the eye is in the primary position. A closely coapted and taut capsule, on the other hand, does not show this elastic movability. Marked

variations occur in this respect even in extreme deviations. An interesting and practical observation is as follows: If the muscle stump is grasped by forceps (after the muscle has been severed and freed) and forcible rotation outward (in case of internal deviation) is made, marked resistance to free rotation may sometimes be noticed. If the capsule is taut and traction on the stump tends to point to its being the cause of obstructed rotation outward, vertical incisions may be made in the capsule extending almost to the lateral borders of the superior and inferior recti (fig. 3). This can be done with impunity when required, if definite scleral fixation is practiced. Even after this vertical capsulotomy is accomplished, forcible traction meets with obstruction to rotation in a few cases. The reasons are hard to fathom when both muscle and capsule are freed. Among speculations as to the cause

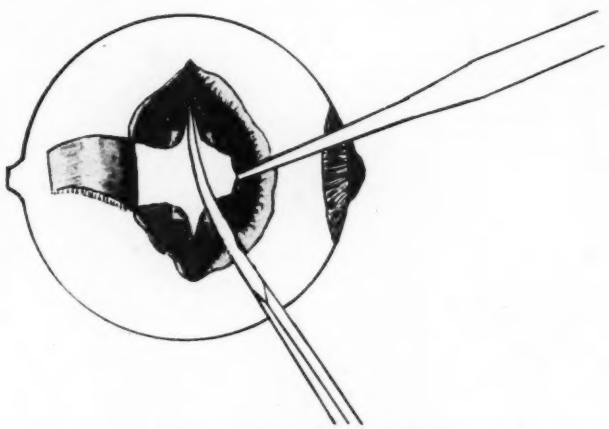

Fig. 3.—Vertical capsulotomy.

of this obstruction to forcible rotation is that of generally contracted capsular or retractive system on the deviating side. There is even the possibility of a short orbital portion of the optic nerve associated with anophthalmos.[3]

I think that one is not exceeding rational reasoning to consider that an eye deflected for many years acquires fascial contractions conforming to the fixed position of the globe in which the capsule is an important part, and it is still within reason that a check ligament not called on to perform its function of a shock deterrent on the opposing side for a number of years may lose some of its elasticity and capability of stretching and, assuming contractility, may act as a deterrent to outward rotation. These may be among the attributable causes of

3. Or what is not unlikely, the suggestion of Dr. James W. White that the check ligaments on the opposing side are the obstructive cause.

unattained operative results in all operations on muscles, so erratic and baffling to the operator after he has made a careful computation and operated to his satisfaction. One can therefore, with limitations, carry the operative plan into the capsular field and thereby augment correction. That these observations are not mere theory or speculation is evidenced by conditions that obstruct muscular function and resist surgical correction, as in the contractive syndrome and the evident relation of muscular action and reaction to these contractions. To pursue the surgical study of fascial contractions further in this direction would, I believe, contribute knowledge that could be utilized in the correction of these obscure conditions.[4]

Much as one can speculate, however, on this observation concerning traction after the muscle has been severed, the one practical point is that a vertical capsulotomy will sometimes release a demonstrably bound capsular system and materially aid in correction as well as in restoring the muscle acting on the opposing side.

MUSCULAR CONDITIONS

Likewise, in viewing the operative field after thorough exposure by turning back the conjunctival flap, one gains valuable knowledge of the physical condition of the muscle: its breadth, length, insertion and attenuation or tension. In no other way can this be intimately obtained. Not infrequently great disparity, sometimes hard to explain, exists in the relation of the physical aspect of musculature to the extent of deviation.

The short tense giant muscle with insertion to the globe abnormally advanced, a muscle that retracts vigorously when severed and when accompanied by a contracted not readily elastic capsule (a combination that I have frequently demonstrated), would require the extreme limit of recession with a possible vertical capsulotomy. Incidentally, it is the type that I feel sure was responsible, when anchorage was not secured, for many of the retracted and inverted caruncles and secondary deviations noticeable in the early days of complete tenotomy; also for the overcorrection and failure of advancement to give the adequate return in correction, and finally for the sudden and disappointing external deviations when the tenotomized muscle has been cut until an effect is obtained by a guarded tenotomy which, in turn, has not been guarded by scleral fixation of the end of the muscle.

Contrariwise, among other instructive findings gained by actual inspection is a lax, sluggish large muscle with a loose capsule which

4. Sir George Berry mentioned a condition that he designated as strabismus fixus, hard to operate on and probably allied to the contractive syndrome type.

makes no effort to retract and, if it were not to preserve anatomic placement, would not require muscle forceps to hold it. Such an anomaly is sometimes found and, although accompanied by a marked internal deviation, if associated with a remote near-point of convergence would greatly reduce computation in millimeters of recession if not contraindicate it altogether. It is hard to understand how with a marked fixed internal deviation a lax, though large and overdeveloped muscle without tension may exist.

I cannot but feel that there must be a change in the cycle of the life history of a muscle as different conditions pertain, and it is possible that in the early history of an accommodative squint, the muscle may be hypertrophied and tense, and as the condition of deviation becomes fixed, relaxation takes place, leaving a large but inactive muscle. This may account for the phenomena accompanying some internal deviations of high degree which give evidence of relative weakness of the interni, with a remote near-point of convergence.

The actual observation of capsular and muscular conditions has been and is an interesting and valuable study in recession. Both these conditions illustrate the value of practicing the entity of recession with its careful inspection of the muscle and capsular field after exposure.

DISSECTION OF MUSCLE AND CAPSULAR FLAP

In the early experimental work on recession the muscle was dissected out in the usual manner, as in preparation for advancement, but as experience ripened, it became evident that an object to be attained was the preservation of muscular power to protect convergence after recession, and a new technic was practiced.

Instead of isolating the muscle and stripping it to its fiber element, which undoubtedly weakened it, I adopted the plan of making a flap retroplantation of the muscle and capsule (fig. 4). This is the reason for the broad flap described in the operation with its diverging incisions.

This procedure is of value as it leaves the muscle in normal relationships and protects it from injury as to its fiber, vessel and nerve supply, which in turn contributes to its ability to conserve convergence after the operation has been performed.

If it is desired that an extended tenotomy be done, the flap is elongated by carrying the lateral incision well back of the equator; the trabeculae between muscle and globe are entirely separated, the capsule on either side is undermined and, if necessary, a vertical capsulotomy is performed.

When a moderate recession is undertaken, these dissections may be modified or abbreviated to suit the exigencies of the case. In either

event they can be performed with a comparative degree of impunity if scleral fixation and gradation are practiced.

To further this same protection of muscular structure, it was found unnecessary to make any dissection posterior to the caruncle other than the separation of the muscle from the globe. The conjunctiva overlying the caruncle should not be separated from it nor should the areolar tissue underneath the conjunctiva be sliced into or disturbed. This, as will be seen later, is unnecessary mutilation.

I abandoned dissection in the subcaruncular or subconjunctival region over the caruncle. I emphasize this, as it has since been advocated, evidently under the erroneous supposition that it is necessary to permit the muscle to slip back under these tissues for readjustment after the recession is performed. Investigation will show that the

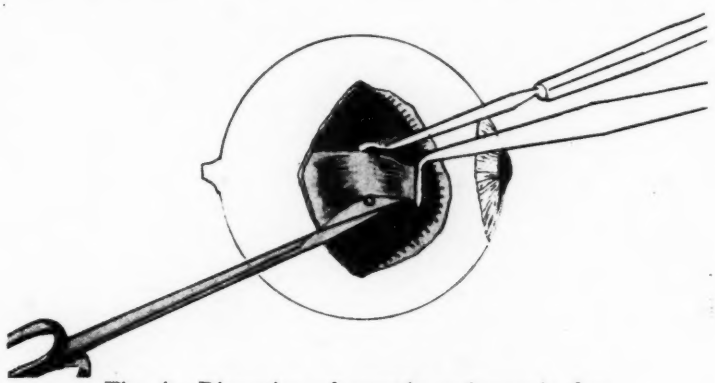

Fig. 4.—Dissection of capsule and muscle flap.

recession of the muscle is principally one of the relation of the muscle to the globe and not to the orbital content or caruncular tissues, and it is doubtful whether, after correction is obtained, the end of the muscle is not on the same plane in relation to the orbital content and caruncular tissues as it was before the operation; for it will be understood that the end of the muscle itself has no opportunity of receding as, being definitely sutured to the sclera, it is pulled outward as the eye rotates outward to correction. While it is true that the end of the muscle has changed its position backward by, say, 5 mm. on the globe, it undoubtedly regains any backward change in its relation to the orbital content by being pulled forward as outward rotation and correction are accomplished. This will be understood by referring to figure 5. The realization of this fact enables one to leave inviolate the tissues in this caruncular region, thus contributing to the conservation and protection of the convergence system, which is so sensitive to surgical intervention. But while the muscle does not require a

pocket dissection under the caruncle to enable it to slip back, the eye requires the opposite provision to enable it to rotate outward, and this is provided for at the outer lips of the lengthy incisions which outline the flap. The ordinary capsule has enough elasticity here to provide for any readjustment that may be required to permit outward rotation; a taut capsule, should it exist, will have been treated by vertical incisions in the manner previously mentioned.

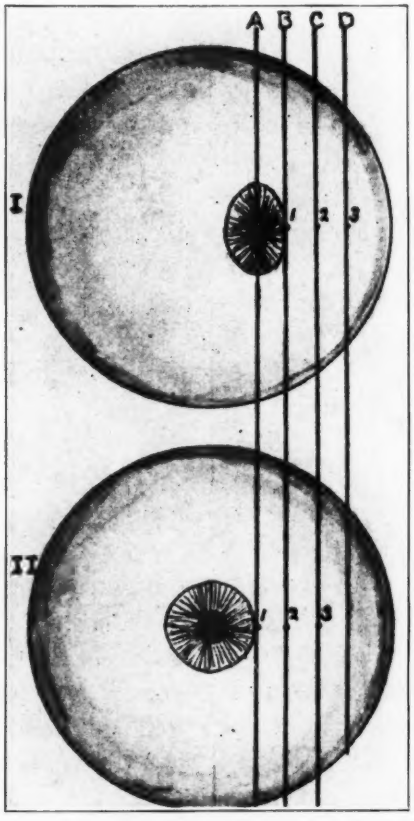

Fig. 5.—Approximate relation of muscle to orbital content before and after correction. *I*, before correction: *1*, corneal margin; *2*, muscle insertion; *3*, muscle recession. (*A*, *B*, *C* and *D* represent fixed lines.) Globe swings around. *II*, after correction: *1*, corneal margin; *2*, muscle insertion; *3*, muscle recession. Before correction, muscle insertion is on line *C*. After correction, muscle recession is on line *C*.

Again the utilization of the flap is the best manner of extending the tenotomy, which means the extension of its corrective capabilities under safely controlled conditions. If the operator attempts, as I did in my first trials, to do a recession without utilizing the diverging incisions on either side of the flap, as is the manner of performing tenotomy,

he will find a limited application. He not only will be cramped for room, but if the capsule should be closely coapted, he will have difficulty in pushing back the end of the muscle. Without the diverging horizontal incisions, readjustment will be faulty and his attachment of the center of the muscle end will be extended in the direction of the cornea. By reason of the difficulty of extending the capsule at the ends of the incision, the line of suturing will, of necessity, tend to describe a curve with the convexity toward the caruncle. The squared-off flap with diverging incisions, however, enables the operator to have a squared-off suture arrangement, and the recession is extended with facility to any desired position with surety of subsequent smooth adjustment of tissue.

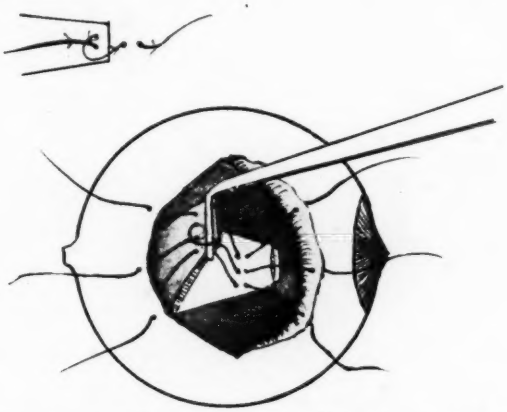

Fig. 6.—Introduction of sutures. Detail of central suture.

ARRANGEMENT OF SUTURES

The aim should be to secure a broad, well spread, properly placed, secure attachment. Single suture operations are not suitable, as they make for a contracted or pointed attachment. The best procedure is the usual one of a single loop with spreading wing sutures. To safeguard the muscle from pulling out, I devised the method of "double looping" a single central suture both above and below: After looping underneath, one passes a hitch on the surface around the ingoing suture, as can be readily understood from figure 6. When the through-and-through sutures are tied (as in the previously described steps of the operation), this suture is involved in a surgical knot which holds a bundle of fibers in a vise that cannot easily pull out. If catgut is used, a looped muscle bundle may be ligated with a surgical knot. The central suture is the mainstay, and the wing sutures spread and attach the angles of the muscles to the desired position. This through-and-through suture arrangement in common use in operations on the

muscles is the best. It facilitates the operation and, if the conjunctival flap has not been faultily dissected (made too short), the closure will be all that is desired (fig. 7).

THE SCLERA AS A HOLDING MEDIUM

It is hardly needful to comment on the suitability of the sclera in the region of the equator as a supportive medium for the pull that a recessed muscle will exert upon the globe. The sclera has been tested out in this manner throughout the history of tenotomy. What had to be established, however, was the strength of the scleral fiber at the point of the attachment as a holding medium for a muscle sutured to it.

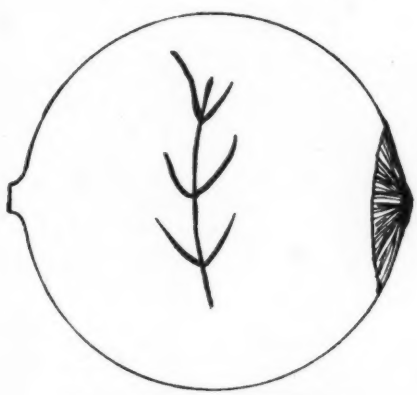

Fig. 7.—Closure of wound.

On a freshly enucleated eye a suture introduced in the sclera at the equator (according to technic presently to be described) was looped, and gradually increasing weight was attached to the suspended thread, over 1½ pounds (680 Gm.) being added before the scleral fiber parted. Moreover, the point at which it finally tore out was clearly confined to the point of entrance and exit of the suture. There was no general laceration of the sclera or of the surrounding tissues, and when the inner surface of the sclera was exposed, the interior was unruptured. If the small bundle of fiber included in the 3 mm. area will hold 1½ pounds, the resistive area to which the muscle is reattached must leave a wide margin of safety from any pull that possibly could be exerted.

SCLERAL SUTURING

Possibly there is nothing that has contributed more toward making recession a workable, acceptable operation than the demonstration of a technic that made the scleral suture feasible, accurate and safe. Again

there is nothing that militated more against its acceptance than the belief that scleral suturing in general, and particularly in the region of the equator, where the sclera is thin, was a dubious and dangerous procedure. This opinion, frequently expressed, has proved to be academic. The technic of scleral suturing, if followed faithfully and intelligently, makes the procedure absolutely safe. I have introduced sutures in this region (splitting the scleral fiber for their reception) for over eleven years and have never had a dangerous reaction or an interior involvement. This is also the experience of many who are now practicing recession.[5] In the light of such experience, safety has been demonstrated.

The technic that I devised is based on simple premises, namely, the observation that a thin lamella of scleral fiber is translucent, and that

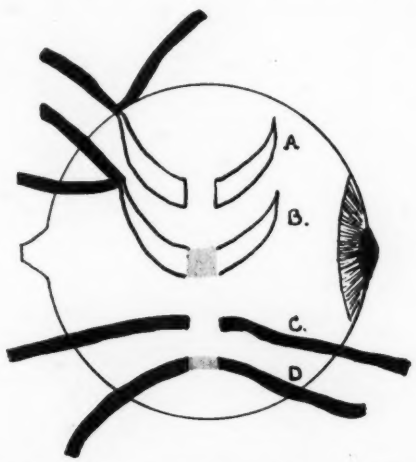

Fig. 8.—Scleral suturing. *A*, needle which has perforated the sclera; *B*, needle which has not perforated the sclera; *C*, suture of perforation; *D*, suture of nonperforation.

a needle or suture passed under such a lamella is always visible from its point of entrance to its exit. Contrariwise, a needle or suture passed through the entire thickness of the sclera is invisible from its point of entrance to its exit (fig. 8).

A little practice on an enucleated eye before scleral suturing is undertaken will demonstrate this and be useful to the operator. Ten or twelve sutures in its circumference will suffice to give him confidence, and inspection of the inner surface will furnish ocular proof of nonperforation.

5. "It is my opinion that the recession operation is less dangerous for a skilled operator because of the fact that a clear view of the sclera is obtained when placing the sutures in position."—Doherty, W. B.: Clinical Reports, New York Eye and Ear Infirmary, March, 1930, vol. 1, no. 1, p. 15.

I cannot do better than quote from my former papers in describing this technic:

> The field and sclera should be sponged of all débris and exposed smooth and white as marble. The needle must be sharp, small, strong and slender, full curved 14 mm. from tip to eye. A large blunt needle will burst and mutilate the fibres and will cramp the operator for room. The needle engages carefully but boldly at first and the point always watched and visible is driven through translucent sclera picking up bundle after bundle of fibres. As the point becomes indistinct it is made to approach the surface but not put through the surface (until exit of needle-point), steady pressure being exerted as it makes its way through the tissue and engages in its bend substantial bundles of fibres. The motion employed is such as one could adopt in picking up the fibres of a heavy linen handkerchief bent over the finger without pricking the underlying finger. The bend of the needle after counter puncture should be visible in its entire continuity through the translucent sclera as also the suture after it is drawn.

Performed after this manner, scleral suturing will hold and can be manipulated with the greatest confidence and safety. With a perforating suture neither the point of the needle, the needle bend nor the suture between entrance and exit can be seen. This technic gives the operator positive understanding that his suture is firmly anchored, which may not be the case if he attempts to suture haphazard. After he has mastered it, he will not have any anxiety in practicing scleral suturing. This visibility of the point of the needle, the bend of the needle and the bend of the suture when it is drawn are infallible guides. They will never fail the operator. They give him confidence and security and provide a safe technic for scleral suturing on any part of the globe.

Needles and Instrumentation.—I had not gone far in the experimental work before feeling the necessity of a suitable needle for scleral suturing or splitting. The scleral fiber being so compact and so closely knit, resistance in the transit of the needle was noticeable. Especially was this seen when the eye with its suture content was reached. A jerk was necessary to start the suture in transit, and this sometimes resulted in a slight tear or laceration at the point of entrance. I therefore devised a needle intended to obviate this, and had it made by P. Moria, of Paris. It is modeled on the following principles (fig. 9): 1. The construction of the blade and the body of the needle should prepare a passageway roomy enough to admit the suture and eye of the needle with ease. 2. The eye should be so constructed that, when threaded, the suture should occupy the least possible space on either side. The first requirement was accomplished by making the blade of fair breadth and by an elevated rib on the surface, passing from tip to eye; the second, by placing the apertures latterly and by engrooving

them, the grooves being prolonged to the butt or end of the needle. Thus the bulkier eye and its suture content are provided passageway by the broad blade which is on the same plane with them, and the lateral engrooving of the sides of the eye and the butt lend still more room and smoother transit of the suture by permitting the suture to insinuate itself latterly well into the body of the needle. The rib is stamped flat in the center of the bend, so that the needle-holder will not turn.

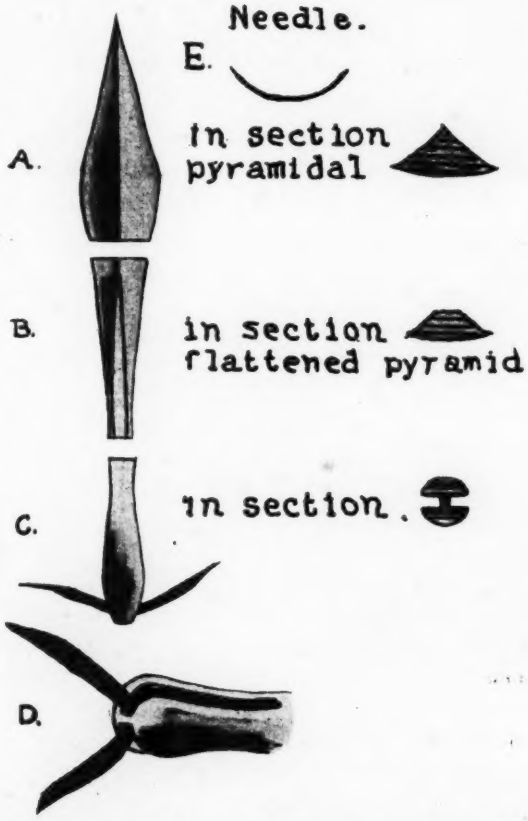

Fig. 9.—Principles of needle: *A*, javelin-shaped cutting blade. *B*, flat saddle so needle will not turn in holder. *C*, needle butt engrooved on side. *D*, detail of butt and engrooved eye. *E*, actual size of needle, 14 mm. from eye to butt, full round.

Other instruments (fig. 10) that seemed necessary and that I devised were the muscle forceps, with teeth on one blade and corresponding perforations on the other, which holds without slipping (it has a 10 mm. rule on the outside of one blade); a strabismus hook which has a fairly large probe point, with a niche behind this point that prevents the muscle from slipping off and a curved recess extending back of the plane of the shank for the reception of the muscle,

and, finally, a modification of a commercial caliper which I shall mention under equatorial measurements.[6]

Suture Material.—In the first hundred cases, no. 1 and 2 twisted silk was used, but in children some difficulty was experienced in the removal of sutures, which were at times hidden by hypergranulations. Trials of different material, principally kangaroo tendon and catgut, resulted in the selection of the latter. Kangaroo tendon is not suitable because of its tendency to swell greatly (to two or three times its caliber) during the period of disintegration. The use of catgut in many operations has convinced me that this is the ideal suture for operations on ocular muscles.

The careful modern preparation of catgut, both chemical and thermal, assures sterilization.[7] Its safety for operations on ocular muscles is almost doubly assured by the fact that danger from the

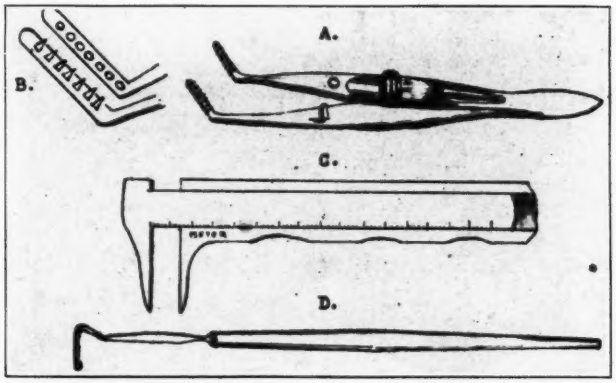

Fig. 10.—*A*, recession muscle forceps. *B*, detail of teeth of forceps. *C*, caliper. *D*, strabismus hook.

spore-forming anaerobic organisms (if they exist) is reduced to the minimum by reason of direct oxygenation, rapid disintegration of the suture and the fact that it can be directly permeated by bactericidal solutions throughout the short period of its retention. If the catgut is not pliable after removal from the vial, it can be made and kept so by being placed between the folds of a moist sterile towel.

6. These needles and instruments may be procured from: P. Moria, 108 Boulevard St. Germain, Paris, France; E. B. Meyerowitz, 520 Fifth Avenue, New York, and Paul Tiemann & Company, 107 East 28th Street, New York.

7. The dissipation of any question of the safety of catgut will be accomplished by those firms which subscribe to the test and standards devised in the Bacteriological Research Laboratory of the Department of Surgery, College of Physicians and Surgeons, Columbia University (see Meleney, Frank L., and Chatfield, Mabel: The Sterility of Cat-Gut in Relation to Hospital Infections, Surg. Gynec. & Obst. **52**:430 [Feb.] 1931).

Until the present time, the finest catgut on the market has been 00, which can well be used, but 000 (made for me) is now procurable for ocular work.[8] This corresponds to no. 1 silk, but as its caliber increases during its sojourn in the wound, it may correspond to a much greater caliber before disintegration. The 000 catgut is chromicized so as to last ten days before disintegration. I use this kind invariably. Its steady use is convincing.

A silk suture becomes more irritating the longer it is retained in an open wound.[9] Catgut becomes less irritating the longer it is retained. Silk of small caliber tends to cut into tissue, especially if it is tied tightly and subjected to tension. On the contrary, catgut, by reason of increasing caliber, cuts less and holds without trauma. This principle is exercised by florists who use raffia in tying the stems of delicate cut flowers.

Catgut, by its faculty of increasing caliber, occupies the channel made by the suture as its surrounding begins to fall away. Moreover, after it has served its purpose, catgut rapidly disintegrates. It lends itself to postoperative sterilization by its permeability and above all the suture does not require removal; for this reason, stitch abscesses from retained sutures, one of the causes of late infection after operation, is avoided and prevented. I allow the patient to be discharged from the hospital after the sixth day without removing the sutures, and have never experienced the slightest trouble.

EQUATORIAL MEASUREMENTS

In early experimental work the question presented itself as to the extent to which recession could be carried out. It was conceivable that under the protection of scleral fixation of the end of the muscle, it might be possible to place the muscle behind the equator if desired; on this supposition the early operations were performed, the establishment of the exact position of the equator being disregarded. Experience, however, taught that this was permissible in only a few selected cases (notably those of short, giant muscles), and also on the external muscles. To achieve conservative work I feel sure that the equator should be the boundary back of which fixation should not go.

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8. This catgut, which complies with the aforementioned tests, may be procured from Davis & Geck, Inc., 217 Duffield Street, Brooklyn.

9. In Engand as early as fifty years ago, Lester noted that catgut was less irritating than silk, and recently it has been recommended as an excellent suture for work on muscles by Mr. J. F. Cunningham (Tr. Ophth. Soc. U. Kingdom **45**:417, 1925).

In the average-sized eye with the average insertion of the muscles, the equator should be situated about 5 mm. back of the insertion of the internal rectus and about 2.5 mm. back of the insertion of the external rectus.[10] These measurements, however, relate to the average-sized eye, and it is known that eyes differ in size. To estimate exactly, a small caliper is essential. I had a modification made of one of the commercial calipers on the market, which fulfils the requirement. To establish the position of the equator in a given case two measurements are necessary: (1) the diameter of the cornea; (2) the distance from the insertion of the muscle to the margin of the cornea.

In taking the first measurements, I had in mind the practice of the rule of Priestley Smith, i. e., that in the average eye the diameter of the cornea is 11 mm., and that a greater diameter indicates a larger globe and a lesser diameter a smaller. Thus, if the corneal diameter measures more than 11 mm., a position slightly more than the average 5 mm. back from the insertion of the muscle would be the location of the equator on the internal side. If the corneal diameter is less than 11 mm. in diameter, a position slightly less than 5 mm. from the insertion of the muscle would be the location of the equator.

The second measurement (the distance between the insertion of the muscle and the corneal margin) to be normal should be 5 mm. for the interni and 6.5 mm. for the externi. If in either instance this is less than the average, in other words, if the insertion is nearer the corneal border, the position of the equator would be more than the average 5 mm. (internal side) and 2.5 mm. (external side) from the insertion, and in the instance of the insertion being further back, a lesser amount than the average would bring the ends of the muscle to the equator.

COMPUTATION OF GRADATION

Notwithstanding the enthusiasm following a series of recessions that were uniform and satisfactory in result, and the wide range of readjustment that a muscle exhibited when definitely fixed at the equator, certain overcorrections and undercorrections, moderate in number, evidenced that the placing of a muscle at the equator as a routine matter indicated carelessness, and that to reduce erratic postoperative results to the lowest degree, the most careful and accurate computation should be made as to the number of millimeters a muscle should be placed from its insertion.

10. I asked Dr. John Evans to establish the approximate position of the equator on the normal eye, which he did on an eye drawn to scale by taking the average measurements obtained from Sweet, Weeks, Dixon, Salmon, Fuchs, Thompson and Whitnell.

Rules for the Amount of Recession.—The guides as to the number of millimeters the muscle should be receded that are in common usage in tenotomy must have some important precautionary additions for recession:

1. The equatorial position (defined in the preceding paragraph) and the number of millimeters the operator desires to recede the muscle should be computed and measured and the points noted.

2. The one principle never to be forgotten is that the muscle should never be receded behind the equator, the position which averages 5 mm. from the insertion of the muscle on the internal side and 2.5 mm. on the external side, except under exceptional circumstances.

3. The actual degree of deviation, fixed, alternate or periodic, bears on the amount of recession. A greater amount would be necessary if the squint was fixed.

4. Marked correction of the deviation with glasses would lessen the amount of recession.

5. The excursive power of the opposing muscle as ascertained by its ability to rotate to the canthi is a valuable indication. If this is diminished, a greater amount of recession is indicated. If the opposing muscle has active rotary power, the amount of recession should be less.

6. The resistance after traction made by a strabismus hook before the muscle is severed is a valuable indication of strength and tension.

7. When exposure yields information that runs counter to the preoperative computation, one can close the wound and proceed on the opposing side.

8. Insufficient convergence with a remote near-point in internal deviation would decrease the recession or contraindicate it altogether.[11] An important point to remember is that a recession on the externi can be freely extended behind the equator if the interni are markedly insufficient, but should be conservative if the interni have active convergence power of 50 mm. or less.

Further, I would call special attention to what I consider one of the most valuable aids and the one that I rely on greatly to obtain a rational understanding of what should be done in an individual case: an actual survey of the anatomic conditions pertaining to both muscle and capsule after thorough exposure by means of a turned back conjunctival flap, as has already been discussed. I feel sure that in the past the operator has missed much by doing a tenotomy "in the dark." A study of the existing muscular, capsular and fibrous conditions obtained by this exposure cannot be overestimated.

11. Dr. James W. White, of New York, recently summarized the indications and contraindications thus: "In a convergent strabismus, if the convergence near point is less than normal, also when the deviation for distance and near are about the same, if the interni are normal but the externi are less than normal, an operation to increase the action of the externi should be chosen. For either a convergent, divergent or vertical anomaly, where a weakening of muscle is indicated, the operation of recession is a more precise and a safer procedure than simple tenotomy" (unpublished lectures on muscle anomalies, 1931).

1. A strong hypertensive muscle with a point of insertion nearer than normal to the corneal margin, when accompanied with a tense, contracted capsule, would indicate a greater amount of recession. 2. A relaxed nontensive muscle with a loose capsule, exhibiting sluggish retractive evidence after tenotomy, would indicate less recession. The findings of which these are illustrations undoubtedly contribute to an understanding as to what operation should be done and the degree of correction that should be sought. They are valuable in making a computation when placed in conjunction with preoperative objective tests.

Another valuable factor in computing the indications and contraindications for recession or advancement and one that enters into the question of balance of both is the tension of the suture. Every operator recognizes the fact that sutures may slip, and the greater the amount of tension on a suture the greater the danger of the slipping or tearing out. The fact should be recognized that advancement always increases the tension of sutures on the advanced side and also on both the advanced and the receded sides if the combined procedure is done. This leads the astute operator not to put an unbalanced amount of advancement on a deviating eye when indications point to the fact that a recession, single or in combination, can be introduced into the operative computation. Attention to this point reduces the tension of sutures to the minimum and makes for the stabilization of the corrective result.

The actual demonstrable muscular and capsular findings should be utilized to obtain the best conclusion. Finally, in making these computations conservatism should be practiced. It is best to err on the side of a slight undercorrection.

Amount of Correction That Can Be Expected from a Given Number of Millimeters.—An estimate of the amount of correction obtainable in degrees must be determined by averages, and exceptions will always be experienced. I find that the clinical amount of correction differs greatly from the mathematical computation.[12] When practicing the extended tenotomy of recession on the internal side I find that it is in fact almost twice the mathematical computation. On the externi, however, the amount of correction obtainable is much less and more nearly corresponds with the mathematical. A single correction of 5 mm. on the internal side will correct a high percentage of from 20 to

12. Mr. G. F. Alexander has pointed out that the mathematical computation of rotation of the globe is 2½ degrees to 1 mm., although he recognizes that this cannot always be taken literally in practice. Campbell, Kenneth E., and Alexander, G. F.: Lancet, June 1, 1912.

25 degrees of deviation. A bilateral recession (internal) of 4 or 5 mm. will correct from 40 to 45 degrees of deviation in a high percentage of cases. Recession of the external muscles, although it can be extended behind the equator, will not give one third of the correction obtained on the interni, bilateral recession correcting only 15 or at most 20 degrees of outward deviation. If the deviation is more than this, it should be accompanied, as a general rule, by an advancement.

I have had little or no experience with hyperphorias, most of which are paretic. I learned last year, however, that Jackson [13] reported two cases twenty-nine years before this writing, in which he receded and sclerally fixed the superior rectus for paralysis of the superior oblique, giving the number of millimeters for that purpose.

POSTOPERATIVE READJUSTMENTS

The absence of secondary or late postoperative deviation is a gratifying feature of recession. I have had under observation some of the early cases (in patients operated on ten years before this writing), and in no instance has it appeared. Several factors have contributed to this: (1) prevention of unnecessary mutilation in dissection of the muscles; (2) definite limitation of the amount of recession, and the establishment of the equator as the definite point back of which the muscle should not be receded; (3) definite anchorage of the muscle by scleral suturing; (4) the selection of suitable cases, and (5) careful computation in gradations.

To one whose experience covers the period of early free tenotomy there will be a striking contrast in muscular action after the recession. In an overcorrection following tenotomy the tendency is to a steady increase. In recession, on the other hand, the tendency is to hold the correction during the healing period and not infrequently to overcome the overcorrection after the muscle has healed and resumed its activity. While the secondary or apparent deviation in the former tended to increase, that of the latter leans toward a decrease.[14] As noted under gradation, a demonstrable insufficiency of muscle on the deviating side would contraindicate anything but a conservative recession.

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13. Jackson, Edward: Operation on the Tendon of the Superior Rectus for Paresis of the Superior Oblique, Ophth. Rev. **22**:61, 1903.

14. A valuable observation by Dr. James W. White is that some such secondary deviations are not attributable nor have they any relation to misplaced anatomy, but are consequent on faulty judgment in doing a tenotomy on the deviating side which careful preoperative examination would have revealed to be insufficient (unpublished lectures on muscle anomalies, 1931).

Moreover, temporary external deviation and outward wandering of the eye during the period of healing is frequently noticed after a well regulated and balanced operation on the muscle and must be attributed, not to overcorrection, but to the fact that the muscle will not function while wounded. When healing is completed, the muscle often immediately functions and parallelism is obtained.

Another gratifying accompaniment of recession is the restoration of function of a weak and seemingly paralyzed muscle on the opposite side. This is frequently noticed in extreme degrees of internal deviation. An externus not capable of abducting to within 5 mm. of external canthus frequently regains its function to normal excursion if not truly paretic. It is particularly noticeable when abnormal muscular development, contracted fasciae and attachment to the globe farther forward than normal exist on the opposing side.

CARUNCULAR RETRACTION

It has been most noticeable that no caruncular retraction or inversion has occurred. This, I feel sure, has been safeguarded against, as has secondary deviation, by recognition of the equator boundary, the negative caruncular dissection, and scleral anchorage of the end of the muscle. I feel confident that the muscle is the beam that gives support to all the tissues in the caruncular region, and that this beam, if secured in the proper place and manner, as practiced in recession, will not permit the caruncle or caruncular tissue to fall away.

Not only the securing of the muscle end but a dissection void of mutilation around the caruncular region should be avoided. This unnecessary mutilation by dissection of the conjunctiva over the region of the internal canthus was recently stressed as a means of preventing caruncular sinking of tissues which in reality it invites. It is advocated under the erroneous impression that the muscle on being receded on the globe must also of necessity recede in relation to the caruncular tissues, which is not the case. Recession, I emphasize again, is only a change of position (after correction) in relation to the globe and not to the orbital content or caruncular tissues. This is one of the strong reasons why caruncular retraction does not occur and why the conjunctiva and tissues in the region of the caruncle should be left unmutilated.

FOLLOW-UP TREATMENT

One of the objects to be attained in the follow-up treatment is the control of reaction and the enhancing of the healing process. Every capsule that is opened and every muscle that is separated from the globe secretes freely, producing an accumulation of serosanguineous

fluid. The secretion accumulates in twenty-four hours and the conversion of the dressing, which it infiltrates, into a moist pad tends to poultice the eye and gives it the nidus and temperature suitable to bacterial growth. A ten or fifteen minute application of ice, a bactericidal solution, such as mercurochrome-220 soluble, 2 per cent, or a silver preparation (collene), 50 per cent, and gentle irrigation with boric acid when the pad is changed (three times daily) will obviate this and keep the field of operation clean while lessening the inflammatory reaction in any operation on the muscles. There will rarely be any chemosis or swelling if this is done, and the reaction in recession is surprisingly little if the operation is smoothly performed.

I early instituted the use of ice for short daily periods, but the recent advocating of almost continuous ice compresses appeals to me as being as absurd as it is impracticable from the nursing standpoint. The healing process should be undisturbed; rest both for the eyes and for the body should be advocated for the first few days. Both eyes should be closed during the early healing period unless exceptional circumstances present themselves. Sometimes if an undercorrection is perceptible, the eye not operated on may be closed and the other eye, with the pad removed, may be induced to fix; or when both eyes have been operated on, the closure may be alternated from day to day. This frequently increases the correction.

The recently advocated routine orthoptic exercises or an endeavor to train the fusion faculty or the eye to fix during the healing period, after swelling has subsided (and in 99 per cent of the cases there is no swelling), most certainly should be deprecated; it may do definite harm. The fusion exercises, by still further receding the muscle, would increase an external deviation if that tendency existed, and for the same reason might induce an external deviation even if the tendency were not present. It is, in addition, an unnecessary and sometimes a painful infliction on the patient.

The operator who adopts this procedure of routine orthoptic exercises may be compared to an orthopedic surgeon who, after carefully suturing a ligament to the periosteum, proceeds to pull it apart again during the healing period, irrespective of conditions that would indicate noninterference.

In this connection, moreover, in the early postoperative period of recession for internal deviation, the result cannot always be accurately foretold, as a deviation or a wandering outward is often noticeable. The wounded muscle will not function or respond and the unwounded muscles will go into spasm to protect it from doing so. It is a revulsion

against nature to endeavor to induce the injured muscle to premature action by means of exercises unless there are special indications. It is fairly obvious that, in the main, the eyes should be at rest during the healing period.

Common sense and sound judgment will guide the operator in the immediate use of exercises which, in much the greater portion of cases, should be left until healing has been accomplished.

ALTERNATE ARRANGEMENTS

When first demonstrating recession, I also taught an alternate arrangement, since discarded for the direct method described in this paper. This has an appeal to those who have been accustomed to anchorage of the stump in advancement. It consisted in the same technic and principles as described in this and my previous papers, with the exception that, instead of tying the end of the muscle to the sclera directly at the point of recession, the sutures were only passed through the sclera and then carried on to the stump to be tied there.

Anchorage of the stump is best performed by making the conjunctival incision directly over the insertion of the muscle, with the adoption of the through-and-through suture arrangement previously described, a central loop suture and two wing sutures to spread the muscle and to provide for broad attachment.

This has some decided disadvantages when compared with the present method. It anchors, but does not definitely secure the muscle to the sclera. The sutures are not tied directly to the point to which the muscle is attached. The globe receives more trauma by anchorage and scleral splitting at the point of recession and the added perforation at the tendon of the stump. The sutures are buried in transit and the reaction is undoubtedly greater.

Under the term muscle retroplacement this arrangement with slightly different sutures was later described twice, in 1924 [15] and again in 1929.[16] The writers adopted my previously published principles and technic of recession. The name retroplacement which they gave to it, however, I do not feel will supplant the more natural one. There is as much reason for making this change from the term recession (the antithesis of advancement) as to change the name of advancement to antroplacement at this date.

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15. Agatson, S. A.: Muscle Retroplacement in Conditions of Squint, Am. J. Ophth. **7**:361, 1924.

16. Berens, C.; Losey, R. R., and Connolly, P. T.: Retroplacement for Strabismus, Am. J. Ophth. **12**:720, 1929.

THE APPLICATION OF RECESSION TO ADVANCEMENT

In former papers I refrained from discussing the application of recession to advancement. The first thing to establish was the single entity of recession and what it could accomplish. When such principles and practice and the knowledge contracted therefrom are understood, they can be applied and utilized with advantage in advancement resection, tuck or kindred operations.

When indications are definite and recession is not contraindicated, I make the single or bilateral recession the primary procedure, because in a large proportion of cases correction will be obtained, obviating the necessity of an accessory operation. One who practices and masters its technic and principles will do fewer advancements. As has already been stated, a bilateral recession performed on the internal muscles will correct a high percentage of these deviations, but there are cases showing the higher degrees of deviations in which the patient will not submit to a bilateral procedure, nor may the operator desire—and it may not be wise—to do a bilateral recession. When, therefore, the operative field must be confined to one eye, what shall be the proportional contribution in millimeters of recession and of advancement in the combined procedure? Some considerations aid one in coming to this understanding.

On internal deviations the recession is performed on the actively tractive and converging side where the musculature leans to tension. It is worth remembering also that the operative field of recession is confined within a small space, 5 mm., and though this space is comparatively small, recession has taught that within its limits a large contribution to correction exists.

Advancement, on the other hand, is performed on the relaxed and passive side where, in place of tension, attenuation is likely to be present. The amount to which an advancement or resection may be carried far exceeds that of recession, but it does not contribute to potential corrective contribution when compared in millimeters to that of recession on the converging side.

Any guide must be relative, but there are certain indications under the different classes of advancement which can be discussed:

Resection with Fixation to the Stump of the Muscle.—Traction by coapting the proposed point of resection to the stump, holding them together and noticing the effect produced on rotation, is helpful.

One guide based on the aforementioned general factors in addition to practical experience, I have found, is that the amount in millimeters of recession should at least be doubled in millimeters of resection. A 5 mm. recession with a 10 mm. resection is likely to correct 40 degrees of internal deviation.

The operator will vary this rule according to conditions found on exposure and the preoperative computation that he has made from subjective symptoms. This guide applies only to resection as performed in operations similar to that of Reese—in which the stump is brought up to the insertion of the muscle—but not to that in which the muscle is advanced and resected beyond the point of insertion, when the added leverage obtained by placing the muscle end so much further anterior to the equator gives greater possibilities of correction.

Advancement in Front of the Stump with Resection.—In my own work I have adopted no absolute rule for recession in conjunction with advancement and resection in front of the insertion of the muscle, regarding this procedure as a powerful supplement to fall back on if occasion should present, always with a mental reservation that each millimeter of advancement and resection in front of the muscle insertion may be met with limitation of motion on the opposite side owing to an advancement so far in front of the equator.

Alexander [17] stated that an advancement of the externus to the corneal margin (6 mm.) and a resection of the tendon (7 mm.) will give a correction of 30 degrees. Meller [18] later independently corroborated this, stating: "A change of 30 degrees in the position of the eye is the most that can be expected from an advancement of one externus." If this is the case, a very moderate recession of 2 or 2.5 mm. would complete the correction in this type of advancement and resection in a deviation of 40 degrees.

The practicability of these computations as guides must necessarily and largely depend on the anatomic and innervational conditions on the opposing side. They would be much in excess of what is required if general laxity of muscle or capsule, or insufficiency of convergence existed with the internal deviation. They would yield comparatively little correction if a strong tense muscle, such as I have previously described, existed.

Simple Advancement in Front of the Stump without Resection.—In 1916, McCurdy,[19] of Kansas, devised an advancement operation without resection, in conjunction with which he receded the opposing muscle and anchored it to the episclera. Although contrary to the rule that he laid down, I believe the proportion of millimeters of

17. Campbell, Kenneth E., and Alexander, G. F.: Operative Treatment of Concomitant Strabismus, Lancet, June 1, 1912.

18. Meller, J.: Ophthalmic Surgery, ed. 3, Philadelphia, P. Blakiston's Son & Company, 1923, p. 142.

19. McCurdy, R. J.: Operative Treatment of Squint, Missouri State M. A. **13**:16, 1916.

advancement should usually exceed the millimeters receded, and it must also be recognized that the rule McCurdy applied to his procedure is impossible as a general adjunct to advancement.

I quote from his paper a rule relative to receding the muscle: "The displacement backward should be the same as the displacement forward," or in other words, the distance the muscle should be receded in millimeters should be equal to that of the advancement. This rule could not apply except when the operative field is confined within the limits of 5 mm. in front of the muscle insertion without resection, as in McCurdy's procedure. For in resection advancements practiced either in front of or behind the insertion, the belly of the muscle is capable of being advanced 10 or even 15 mm. to the corneal margin, and to recede to a point 15 mm. behind the insertion on the side of the recession would put the muscle completely out of commission. The operator should therefore rely on the principles of the single entity as described throughout this paper for the number of millimeters which, in his judgment, will best conform to the needs of his case.

Advancement in Paresis of the Lateral Muscles.—There is one observation that I should like to make in regard to the utilization of the entity of recession in conjunction with advancement in paresis of the lateral muscles. An advancement on the paretic side (externus) often has to be carried to the induction of an anchorage between the sclerocorneal margin and the outer canthus. A very free tenotomy is usually done on the deviating side, and even here recession with scleral fixation is an advantage. The end of the muscle can be placed behind the equator, if desired, and may prevent by restraint what I have seen, an unsightly bulging of the eye on the deviating side due to a muscle which, having lost all contact with the globe, has also lost control as an agent of balance.

Comment.—In contemplating advancement, then, unless there are indications why the deviating side should not be explored surgically, I feel that the operator loses much of the stabilization of advancement if he does not primarily make himself cognizant of the anatomic findings, regulating the recession as conditions prevail on the hypertensive side. This can best be done by a thorough exposure of the muscular field.

END-RESULTS IN THE ENTITY RECESSION

Recession is not an infallible operation. No operation on a muscle is. There are many factors that militate against the correction obtainable from any operation on the muscles, and a certain number of failures will always be experienced. Questions of innervation, central fusion influences, contractions of fascial tissue and insertional and struc-

tural conditions of the muscles contribute toward leaving a postoperative residue of noncorrection which cannot be estimated or eliminated and must be remedied later.

The regularity and uniformity of the correction obtained by recession on internal deviations in suitable cases is strikingly large, at least 90 per cent. This is corroborated by many who are now actively practicing recession.[20]

The residue of uncorrection will be about 10 per cent after operation. In this 10 per cent there will be more undercorrections than overcorrections if the operator has been conservative. I regard as successes cases in which one remedies the deviation, leaving not more than a few degrees of actual uncorrected residue with good converging power and unimpaired rotation.

As previously mentioned, one of the most gratifying and sometimes unexpected results in the practice of recession is the manner in which, after operation on a marked internal deviation, an externus that faltered in outward rotation 4 or 5 mm. from the outer canthus regains its function of activity and rotation.

In amblyopic eyes with marked squint (cosmetic) corrections have been counted among successes.

Slight overcorrection is likely to be overcome when the healing process is completed and the eyes begin to function actively, and, as has been mentioned, no secondary deviation has occurred.

GENERAL OBSERVATIONS ON RECESSION

The contributions that recession has made to muscle surgery and the lessons that it conveys, I feel, are as follows:

1. It has demonstrated that scleral suturing in the region of the equator done under proper technical conditions is a safe procedure.

2. With few exceptions the equator is the definite point back of which the muscle should not be receded.

3. Tenotomy can be extended in the practice of recession within safe limits.

4. Recession (with the technic advocated) reduces the possibility of caruncular retraction secondary deviation to the minimum.

5. A high and safe degree of correction (at least 25 degrees) is obtainable by a 5 mm. recession.

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20. For instance, W. Thornwall Davis (in his discussion of this paper when it was read before the Ophthalmological Section, New York Academy of Medicine, March 16, 1931) in this country, and J. Harrison Butler (in discussion of Jameson [footnote 2]) in England.

6. The correction obtainable on external deviations is much more limited and usually requires a supplementary advancement.

7. Muscle function and rotation on the attenuated side is frequently restored by recession on the opposing side if the muscle is not paretic.

8. One of the most valuable aids to computation is the actual inspection and study of muscle and capsule after exposure.

9. The secondary procedure of vertical capsulotomy in some instances extends the effect of correction, and this procedure can be conservatively done if scleral suturing is practiced.

10. It is as logical to regulate surgically a contracted capsular system on the tense side as to do so on the side of relaxation.

11. The clinical amount of correction obtainable by recession in internal deviations is greatly in excess of the mathematical amount as computed in degrees; in external deviations it is about the same as that computed mathematically.

12. Recession makes the best and safest guarded tenotomy.

13. Conservative recession paves the way to safer and better advancements when used in conjunction with them.

14. A rule that seldom can be broken is that if a 5 mm. recession (internal) will not correct the deviation, the further corrective contribution must, of necessity, be brought about by advancement or recession on the opposing side.

15. The single entity of recession gives a notably high return of correction as a primary procedure.

CONCLUSION

The object in this paper has not been to exalt unduly the entity of recession over the antithetic operation of advancement and its kindred procedures, a controversial question that I have been anxious to avoid (realizing that the two must often necessarily go together). Advancement and its kindred procedures require no further brief, and certainly no depreciation when the services that they have rendered and will concontinue to render to muscle surgery are reviewed. My object has been to make known the hitherto unknown corrective values and capabilities of the single entity of recession when the operative technic and principles of the procedure are properly practiced.

I think that it will be agreed that the entity of recession with its principles herein presented, is far more than an anchorage operation and imparts to operations on the muscles a certain much needed security; and that when the safety of scleral anchorage, the limitations as well as the extensions to which tenotomy can be carried, the accuracy

of gradation, and the revelation of the anatomy as the result of dissection are known and practiced, tenotomy—which lends itself to floating attachments or ungraded or unguarded control—will be practiced in fewer cases.

The point as to whether a contractive deviation is best corrected by placing the emphasis of operative correction on the weak and often relaxed and attenuated side as in advancement or on the strong and contractile side with its incident structural hypertrophies and insertional anomalies, has always been a matter of contention. The erratic results obtained in tenotomy with its remote secondary deviations undoubtedly swung or biased the operator in favor of advancement, but recession with its highly protected and large corrective return shows evidence of bringing back the operative trend and making the primary procedure a surgical adjustment on the side of tension, and it is fast overcoming the stumbling-block that confronts every new procedure, that of traditional usage.

Finally, the operator who practices recession will find a combination of much practicability. He will substitute definite scleral fixation of the muscle, obviating the risk of an indefinite floating attachment. He will avail himself of the accuracy that computed gradation contributes toward the attainment of conditions that he desires to bring about.

He will be able to extend greatly, under safe conditions, the correction hitherto unattainable. He will experience the value of operating on a freely exposed operative field instead of groping in anatomy that is obscured and hidden.

He will have the satisfaction of knowing and recording accurately where and how his muscle is attached to the globe in the event of future operative intervention becoming necessary. He will sometimes greatly contribute to the effect of the operation by the adjustment of fibrous or fascial contractions or retractions.

He will experience the assurance and satisfaction of practicing scleral suturing in the region of the equator under safe and guarded conditions. He will safeguard the dissection in the manner best calculated to protect convergence. He will be reasonably sure that no secondary deviation can occur.

He will have a much enlarged and more accurate understanding of what tenotomy should and can accomplish when the principles of recession are applied. He will realize that tenotomy can and has been converted into what it has never been in the past, a safe and thoroughly scientific operation, and that the entity of recession changes the whole complexion of tenotomy, not only as to safety of procedure, but also as to range of correction; and what is true of tenotomy applies also in great

measure to the stabilization of advancement, for one can grade and sclerally fix the recession to conform to the requirements of the advancement, and grade and fix the advancement to the requirements of the recession.

The surgeon who by faithful practice acquires the technic will not be disappointed and will add to the operative procedure that he employs for the correction of strabismus one that I feel sure will reward him with a fruitful and extended return of corrections.

LESIONS OF THE CORNEA AND CONJUNCTIVA IN ERYTHEMA EXUDATIVUM MULTIFORME (HEBRA)

REPORT OF THREE CASES WITH GRAVE OCULAR SEQUELAE *

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This paper is based on three cases of erythema exudativum multiforme in which lesions of the cornea and conjunctiva appeared. I had the opportunity of studying two of the cases in extenso; the third case, details of which are herein appended, was called to my attention by Dr. Benjamin Newman of Brooklyn. The patients presented ocular manifestations that were not isolated phenomena but an integral part of a clinical picture possessing all the attributes of a distinct systemic disease that challenged definite classification. Stevens and Johnson,[1] in an article on a new eruptive fever with stomatitis and conjunctivitis, stated that a search of the literature failed to reveal identical cases, and they considered the possibility of a new disease that hitherto has not been described.

The features of this disease are: (1) an onset characteristic of an acute infection, namely, chilly sensations, fever, headaches, loss of appetite, general body discomfort, and, frequently, pain in the joints; (2) marked inflammatory changes in the conjunctivae and corneas eventuating in serious visual impairment and often in total destruction of the bulb; (3) an intense pseudomembranous inflammation of the mucous surfaces of the lips, tongue, mouth, palate, oropharynx and occasionally of the nares, larynx, bronchi and even the vagina; (4) a widespread polymorphous eruption of the skin; (5) a leukocyte count that may vary slightly in either direction, i. e., indicating either mild leukocytosis or leukopenia, and (6) a protracted course extending into weeks or months. The prognosis of the disease itself, quoad vitam, is favorable, but the ocular sequelae are often disastrous.

It is probable that this disease is not so rare, but escapes detection or is confused with some other entity because the attending physician is unfamiliar with it. The disease offers an ensemble that is unmistakable, and is readily recognized if it is only borne in mind. It is of interest to note that when the condition appeared in children, it was almost invariably diagnosed as hemorrhagic measles.

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* Submitted for publication, March 28, 1931.

1. Stevens, A. M., and Johnson, F. C.: New Eruptive Fever Associated with Stomatitis and Ophthalmia, Am. J. Dis. Child. **24**:526 (Dec.) 1922.

REPORT OF CASES

CASE 1.—H. D., Jewish, a house painter, aged 39, married, was admitted to the hospital obviously very ill. There were no hereditary taints and no history of saturnism. The personal and past histories had no bearing on the case. On Dec. 25, 1927, the patient was taken acutely ill with coryza and sore throat; his temperature was 104 F. The next day he began to cough; there was some difficulty with breathing, and pain in the limbs and joints, general bodily discomfort and a high fever were present. The illness was diagnosed as grip, and antistreptococcus serum was injected. The patient could not retain any nourishment, and a generalized eruption of the skin was noted. He complained of pain in the eyes and photophobia; there was a free conjunctival discharge. His condition became progressively worse, and on the fifth day of the illness he was hospitalized. The following is a digest of the hospital record:

The patient had a widespread eruption of the skin, the individual efflorescences varying much in size and appearance. Some of the lesions were macular, others papular, but most of them were petechial. They were discrete, with normal skin intervening. They measured from 1 to several mm. in diameter, and varied from a bright red to a dusky blue. The rash was profuse, and involved the face, chest, trunk and limbs. The eyelids were swollen, their margins covered with thickened pus, and the cilia were matted; the eyes were held tightly closed. The bulbs were very sensitive and painful. The patient complained bitterly of photophobia and a burning sensation. On separating the lids, which required the use of retractors, the eyeball was found to be smeared with mucopus. The palpebral conjunctivae were tumid, the follicles were prominent, and small scattered areas of necrosis were present. The bulbar conjunctivae were injected, and small necrotic patches were found here also; in addition they were chemotic and overhung a large portion of the corneas. There was a copious mucopurulent secretion containing many strands of fibrin. The corneas were intact. There was, however, marked vascularization of the limbi. The pupils were moderately contracted, and responded adequately in their several reactions. The iridic structures appeared normal. The anterior chambers were not shallow, and the tension was not raised. Examination of the fundus did not reveal a pathologic process. The patient averred that his sight was impaired. The lips were dry, cracked, bleeding and crusted. When the mouth was opened, the buccal mucous membrane was seen to be flecked with ulcers, hemorrhagic spots and fibrinous plaques. The palate, uvula and oropharynx presented a similar picture. The tongue was swollen and beefy and covered with numerous small, dirty grayish membranes. There was an extremely foul odor from the mouth. The cervical glands were enlarged and tender.

The symptoms and signs increased in severity. The ocular, buccal and cutaneous pathologic processes were more pronounced. The mouth was now one mass of ill-smelling pseudomembrane, disintegrating in spots and exposing bleeding necrotic areas. The temperature continued to be high; prostration was marked. It was evident that we were confronted with a serious disease, the diagnosis of which was, at best, a matter of conjecture. Smears and cultures from the mouth and eyes showed the micro-organisms usually found in these situations. Repeated blood cultures were negative. The number of leukocytes was not increased; if anything, there was a tendency to leukopenia. The cytology was normal. Chemical examination of the blood and many urinalyses gave essentially negative results. The eyes were becoming distinctly worse; the ocular pain and tenderness increased, the photophobia was distressing, the conjunctivae presented many necrotic spots, the discharge was more profuse, and several thin fibrinous bands joined the raw surfaces of the scleral and palpebral conjunctivae, forming an irregular network in both the upper and lower fornices.

The left eye was more affected than the right. In the center of the left cornea was a small ulcer; just within the limbus was a deep-seated, annular infiltration resembling closely a ring abscess, which, however, at no time showed a tendency to break down, and was replaced, eventually, by fibrous tissue. There was intense ciliary injection, the eye was very sensitive to light and was held tightly closed. The central corneal ulcer increased in depth and in size, but perforation, although threatened, did not occur. The peripheral opacity continued to spread, and finally included the limbus. There remained, however, a relatively clear space between the central and outer areas of opacification. The pupil was now contracted, irregular and bound down at intervals to the anterior capsule of the lens. The iris was swollen and dull; the markings were indistinct. The intra-ocular tension was not increased. The sight was much impaired.

The right eye suffered much less. Although the palpebral and bulbar conjunctivae were considerably damaged, the cornea was only slightly involved, and no ulceration occurred. There must have been decided iridic irritation; for, in spite of the liberal employment of atropine, the pupil would not dilate to any appreciable extent. The eyeground was normal.

For three weeks the general clinical manifestations did not abate. At the end of this period, improvement was noticed. The temperature soon became normal, the prostration was less marked, the mouth, tongue and throat were more comfortable, the eruption of the skin was fading, and desquamation was taking place, especially on the palms and soles where large layers of necrotic epidermis could be peeled off. The ocular status, however, was a matter of grave concern. The patient stayed in the hospital six weeks.

Several weeks after the patient's discharge from the hospital, the following observations were made. The mouth and skin were normal; there were no scars or pigmented areas. A peculiar phenomenon was that the nails of all the digits of both hands and both feet fell off and were later completely reformed. The patient's general health was good. The eyelids were heavy and drooped, the skin was redundant and loose and exhibited many fine wrinkles, approaching a state of blepharochalasis. The interpalpebral spaces were much narrowed; the right palpebral fissure being 5 mm. wide, the left 3 mm. There was a reflex spasm of each orbicularis oculi accompanied by vertical wrinkling of the forehead, due to photophobia and pain ascribable to a persistent irritating discharge acting in the manner of a foreign body. When this secretion was removed, the eye felt much more comfortable, and could be opened fairly well. One now ascertained that the upper lid drooped—a mechanical ptosis caused by the increased weight of the lid from the presence of adventitious tissue resulting from the intense inflammation early in the disease, a ptosis comparable to that seen in chronic trachoma. In fact, the patient's palpebral conjunctivae simulated the picture found in the cicatricial state of trachoma. The margins of the lids were hypertrophied and irregular, and studded with small scars, the sequelae of numerous discrete ulcers. In the posterior lip of the border of each lid, arranged regularly, were dots of yellowish secretion occluding the outlets of the meibomian glands. Parenthetically, it may be stated, these dots offered a good anatomic demonstration of the number and situation of the ostia of these glands. There was a considerable amount of stringy, tenacious, mucofibrinous discharge, closely resembling that found in severe cases of vernal catarrh. The eyeball and the apposing surfaces of the lids were streaked with a greasy foamy substance that did not admix with the tears, thus preventing the latter from coming in contact with globe. The tears themselves reached the conjunctival sac in scant quantity, owing to fornical scarring. When drops were instilled, they ran down the eyes, as occurs when the conjunctival

sac contains oil or an ointment. The eyes were glassy and besmeared, reminding one of the eyes of a cadaver that has lain for some time in the morgue. The palpebral conjuntivae were dusky. There was abundant scar formation. Both the upper and lower fornices were shallow, and many fibrous bands connected the conjunctiva of the lid to the bulb. The ocular conjunctivae were injected, and the episcleral vessels overfilled. In places, the relaxed conjunctiva overhung the limbus.

There was moderate pain and tenderness in the upper lateral aspect of the right eyeball. The cornea was dull; its luster was completely gone. Peripherally, there was a rather superficial opacity, somewhat circular in outline, and extending unequally toward the center. This opacity contained many fine blood vessels springing from the limbus and converging centripetally. The circumcorneal injection was intense. With a magnification of 10, the whole surface of the cornea was seen stippled with a dustlike superficial infiltrate. Although atropine had been used freely, the pupil was only 3 mm. in diameter, but quite round. The anterior chamber was of ordinary depth; the tension was normal. The iris was somewhat swollen, the markings only slightly indistinct, and the iridic sheen was reduced. There were no synechiae. The eyeground appeared reddened and hazy; this was not to be attributed to the pathologic process in the fundus, but was obviously accounted for by viewing the eyeground through a cornea of impaired transparency. Vision was 20/100; there was no improvement with glasses.

The left cornea was moderately injected at the ciliary border. In the periphery, there was an irregular, annular, superficial, vascularized opacity. Central to this was a clear circular space varying in diameter from 1 to 2 mm., with vessels traversing it from the limbus. The remaining portion of the cornea was opaque. This opacity was heavy, and included almost the whole thickness of the cornea. It was densely white, particularly at the center. It was richly vascularized, and occupied the site of the former corneal ulcer. The anterior chamber was abnormally deep, owing to keratectasia resulting from a diseased cornea yielding to the intra-ocular pressure. The pupil was contracted and irregular, and bound down to the capsule of the lens. The tension was not increased. The iris was swollen, its markings hazy and its polish greatly diminished; several posterior synechiae were present. The details of the fundus could not be seen. Vision was eccentric and limited to counting fingers at 3 feet, and only in the temporal field.

At the time this report was written, three years since the onset of the illness, the patient was practically blind, which was due chiefly to corneal opacification. It is hoped that an optical iridectomy at some future time will give him some vision. The blepharospasm has not abated; the patient keeps his eyes completely closed, and complains of intense burning. The stringy mucoid discharge is unchanged as to quantity and character.

CASE 2.—M. L., a boy, Jewish, aged 9 years, had had pneumonia, mumps, whooping cough, chickenpox, and frequent colds; tonsillectomy had been performed seven years before the present examination. The family history was unimportant.

On Aug. 30, 1927, the child was taken ill with sore throat, redness of the eyes, headache, and a fever of 102 F. There was some cough. The next day the eyes and throat became worse, and a generalized eruption of the skin appeared. The temperature continued to be elevated. The cervical glands were swollen and tender. The child remained at home five days, during which time his condition became aggravated. On the sixth day of the illness, he was hospitalized. The following notes are excerpts from the hospital chart:

The patient was acutely ill and toxic. The eyes were swollen and opened with difficulty; there was a copious, purulent, conjunctival discharge. The mouth, tongue and fauces were covered with mucopurulent exudates; many raw, bleeding areas were present. The cervical glands were enlarged. The lungs were normal, except for roughened breath sounds and occasional crepitant râles. The entire body was covered with a widespread but discrete rash; there were numerous macules and papules, and many hemorrhagic spots. Here and there several umbilicated vesicles were found. The individual lesions of the skin varied much in size, some being 2 mm. in diameter; others were much larger. On September 7, the ninth day of the illness, the record states that the cervical adenitis was more marked; the rash was more pronounced, and was multiform in character (macular, papular, petechial, and vesicular); the eyelids were red and edematous, and there was a profuse, fibrinopurulent conjunctival discharge. When the resisting lids were separated with retractors, there was a soft fibrinous membrane hanging loosely in each eye and attached above and below to the superior and inferior fornices, respectively, concealing the whole anterior segment of the eyeball, forming a kind of curtain that shut the eyeball from view. When traction was made on this membrane, partial separation occurred, and there was free bleeding at the points of severance. The membrane was not adherent to the cornea; it was readily lifted up with forceps, easily torn through, and the subjacent cornea found intact. The bulbar conjunctiva was hyperemic. The palpebral conjunctiva was raw and granular, exhibited a number of necrotic and bleeding spots. There was no further ocular pathologic process. The lips were cracked and crusted with fibrinosanguineous exudate. The tongue was excoriated. The mucous membrane of the mouth and pharynx displayed many hemorrhagic, ulcerous and pseudomembraneous areas which showed a tendency to become confluent. The eyes, oropharynx and skin remained in statu quo for about two weeks. The temperature continued to be elevated; there was much prostration. Then improvement began, and the patient was discharged from the hospital one month after admission. In the hospital, repeated examinations of the urine and blood, including blood cultures, gave negative results. There was no leukocytosis, and the cytology was normal. Many smears from the conjunctivae and mouth revealed mostly *Staphylococcus pyogenes-aureus*. The conjunctival pseudomembrane gradually disintegrated and disappeared, followed by moderate cicatrization. There was no desquamation of the skin, and the mucous membranes healed without scar formation. The subsequent ocular history is of interest. The child persisted in keeping the eyes continuously closed; there was marked photophobia, the corneas became hazy, and ulcers formed, leaving behind dense opacities that caused pronounced visual impairment. Ophthalmoscopic examination, which was quite difficult, showed apparently normal eyegrounds. The apposing conjunctival surfaces were scarred. Many fibrous bands had formed so that the fornices were practically obliterated. The posterior lip of the border of each lid was dotted with inspissated yellow material, indicating the position of the meibomian ostia. The child's vision now is so poor that he is an inmate of a home for the blind. The mother informs me, however, that there is some useful sight in one eye, but hardly any in the other. The lids droop; the child keeps his eyes tightly closed, and is unable to open them beyond a narrow slit.

Case 3.—An Italian boy, aged 7 years, was admitted to a hospital for contagious diseases with the diagnosis of measles. The child had had measles two years previously. The rest of the anamnesis had no bearing on the case.

Six days prior to entering the hospital, the child was taken suddenly ill with a temperature of 106 F. He went to bed complaining of general discomfort and

dizziness. On the third day of the illness, mention is made of pain in the eyes and disturbance of vision. While at home, the temperature varied between 99 and 102 F., there was a discrete eruption on the skin, pain in the extremities, and the patient desired to be let alone.

Physical examination on admission to the hospital showed a moderately well nourished boy, acutely ill, with a temperature of 105 F. The skin was covered with a generalized rash consisting of macules, papules and petechiae. The lids of both eyes were quite swollen, there was a purulent discharge, and the conjunctivae were intensely congested and chemotic. Circumscribed hemorrhages were present beneath the bulbar conjunctivae. There was marked ciliary injection, and the corneas were hazy. The aqueous chambers contained an abundant cloudy fluid. There was a fibrinous exudate on the anterior surface of each iris. The left pupil was regular; the right pupillary area was obscured by inflammatory products. Photophobia was marked. The tongue, mouth and throat were sore, causing the patient to refrain from food. The heart and lungs were normal; the liver and spleen were not enlarged. Cultures from the eyes, mouth and throat were negative for diphtheria. The blood was sterile. The leukocyte count was 16,000 with 72 per cent polymorphonuclears, 19 per cent small mononuclears, 7 per cent large mononuclears and 2 per cent eosinophils. The hemoglobin was 75 per cent. A number of urinalyses gave negative results. The chemical constituents of the blood and the spinal fluid were normal. Koplik's spots were absent.

During the patient's stay in the hospital, the temperature ranged from 100 to 105 F. The edema of the eyelids gradually subsided; the subconjunctival hemorrhages were absorbed; but the cloudy corneas, the turbid anterior chambers, the iridic deposits, the photophobia and impaired vision persisted. On the eighth day, the cutaneous eruption was still present but fading. When the rash had completely disappeared, several large pigmented areas remained on the thighs and buttocks. The patient left the hospital, on release, eighteen days after admission. At this time, the following notation was made: both corneas hazy and, in addition, an annular opacification just within each limbus; anterior synechiae, and greatly reduced vision. I have been informed that both eyes were subsequently operated on without benefit.

A recent examination showed that the boy is practically blind, vision being limited to eccentric perception of light. The conjunctival sacs are smooth and regular; adhesions were not seen between the palpebral and bulbar conjunctivae, and there was no discharge nor evidence of any pathologic process affecting these membranes. There was some circumcorneal injection. The eyeballs were tender, and the tension was rather low. Almost the entire cornea of each eye was the seat of dense opacification, containing many calcified particles. There was no trace of previous corneal perforation. Peripherally, there were a few small transparent areas, through which the subjacent iris, thickened and lusterless, was visible. The anterior chambers were irregularly shallow, and anterior synechiae were present. Photophobia was decided and annoying.

REVIEW OF THE LITERATURE

Hebra[2] (1866) separated from the heterogeneous group of polymorphous erythemas a clinical entity to which he gave the name erythema exudativum multiforme, which he considered due to a systemic invasion by some unknown factor and not caused by a local disturbance. He said:

2. von Hebra, F.: Diseases of the Skin, London, New Sydenham Society, 1866, vol. 1.

Erythema exudativum multiforme is an acute circumscribed inflammation of the skin, confined almost exclusively to the papillary layer. Occasionally, the mucous surfaces are affected. There appear on the extensor aspects of the body and on the face, symmetrically arranged spots, varying in hue from bright red to purple. The efflorescence is noticed quite suddenly, and lasts about two weeks. In the further evolution of the pathology, the spots may become raised, forming papules, or, if the exudate increases and much serum is poured out and the epidermis is elevated, vesicles result. The lesions may assume various shapes and appearances. When they manifest themselves as concentric reddish rings surmounted by an annular vesicular rash between these rings, the term herpes iris is applied. Herpes iris may be the sole efflorescence, or may be present associated with many other types of skin lesions.

Kaposi [3] (1893) gave the following description of the disease:

Erythema exudativum multiforme (Hebra) appears simultaneously and symmetrically on the extensor surfaces of the hands and feet and on the adjoining parts of the arms and legs, with the eruption of pinhead-sized, bright red spots which pale on pressure, which quickly increase in size, are sharply circumscribed, which to the touch are firm and usually edematous, and moderately raised above the surrounding skin. The center of the lesion sinks in quickly, becomes cyanotic or hemorrhagic, while the more recent part presents a bright red area. Gradually there follow new lesions on the face, neck, chest, thighs and back. At times, when the exudate increases, vesicles form. The center falls in through absorption of the liquid contents, the cyanosis or hemorrhages become more visible, peripherally there is a progressive formation of a raised, red, steep, firm margin on which is located a ring of vesicles (erythema vesiculosum). Frequently, one finds about an older center a series of concentric circles of vesicles (herpes iris). The individual lesions disappear, leaving behind a brown pigmentation. Efflorescences with hemorrhagic centers, when retrogressing, assume, sequentially, the colors blue, yellow, green and brown. When a vesicular eruption occurs, it eventually dries up, with the formation of scabs, and desquamates. It is a self-limited disease, lasting from two to six weeks at the utmost. If the eruption involves the whole body and recurs, it may last months or even years. With the appearance of the skin eruption, there may be seen similar lesions of reddish spots, papules and vesicles on the mucous membranes of the mouth and throat, over which the epithelium becomes rapidly turbid and is elevated in the form of membranes and shreds, so that painful wounds arise. Rarely, the epiglottis, larynx, vulvovaginal and urethral mucous membranes are attacked.

Mracek [4] (1902) divided the erythemas into four classes, one of which he designated erythema exudativum multiforme, following Hebra's nomenclature; he considered this condition infectious, although the specific organism has not been identified.

Besides the skin, in many instances, the mucous membranes are also attacked: upon the lips, the soft and hard palates, the tonsils, upon the genitalia, one finds manifestations of the erythema corresponding to the type that appears on the skin, except that in the bullous type you only seldom come across vesicles or vesicle-

3. Kaposi, M.: Pathologie und Therapie der Hautkrankheiten, ed. 4, Vienna, Urban & Schwarzenberg, 1893.

4. Mracek, F.: Handbuch der Hautkrankheiten, Vienna, Alfred Hölder, 1902, vol. 1, p. 540.

formation. The epithelium is too loose to permit it to be raised by a collection of serous fluid, and one finds here only superficial erosions surrounded by a red area with a purulent base. . . . Many authors allude to a peculiar conjunctivitis. . . . The above mentioned manifestations of the skin and mucous membranes are preceded by prodromal symptoms which mostly stand in direct relationship to the severity of the course of the affection. These are weakness, headache, joint-pains, and especially fever . . . that may rise more or less considerably . . . and continue several weeks.

Crocker [5] (1903), in his book on diseases of the skin, referred to this condition as follows:

The onset of the eruption is usually preceded and accompanied by constitutional symptoms . . . sometimes of considerable severity . . . the eruption is very variable . . . even commencing in the mucous membranes of the eye, tongue, and mouth . . . many writers both in France and Germany regard it as an acute specific disease . . . they are worthy of consideration.

Stelwagon [6] (1904) said:

In some cases there may be a good deal of febrile action and often with accompanying swelling and pain about the joints . . . also anorexia, digestive disturbances and malaise. . . . The observation has been occasionally made that . . . the cutaneous lesions are preceded by an inflamed or eruptive condition of the fauces or . . . a mild conjunctivitis . . . the lips and mouth show vesicles or blebs. . . . The most severe types . . . are due to constitutional infection. Indeed this view is gaining ground.

Schamberg [7] (1915) made the following statement:

At times the mucous surfaces of the eyelids, nose, mouth, and throat become involved.

Darier [8] (1920) referred to the disease thus:

Under the name of multiform or polymorphous exudative erythema, Hebra distinguishes in the heterogeneous group of erythema, a syndrome which one is almost tempted to regard as a disease. In its behavior it approaches the eruptive fevers. . . . The eruption . . . is often accompanied by systemic phenomena.

Macleod's [9] (1921) textbook contains the following description:

The mucous membranes are affected in a considerable number of cases, and sometimes more severely than the skin. The mouth and pharynx are most commonly involved. In some cases the mucous membranes of the nose, conjunctiva and genitalia are attacked. The patient may be seriously ill.

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5. Crocker, H. R.: Diseases of the Skin, ed. 3, Philadelphia, P. Blakiston's Son & Company, 1903.

6. Stelwagon, H. W.: Treatise on Diseases of the Skin, ed. 3, Philadelphia, W. B. Saunders Company, 1904, p. 147.

7. Schamberg, J. F.: Diseases of the Skin and Eruptive Fever, ed. 3, Philadelphia, W. B. Saunders & Company, 1915.

8. Darier, J. A.: Textbook of Dermatology, Philadelphia, Lea & Febiger, 1920.

9. Macleod, J. M. H.: Diseases of the Skin, New York, Paul Hoeber, Inc., 1921.

Ormsby [10] (1921) made this statement concerning the disease:

It occurs . . . but rarely upon the mucous membranes of the nose, mouth and conjunctiva . . . and of unknown etiology . . . frequent association with rheumatism and rheumatoid pains, febrile phenomena, malaise, and other constitutional disturbances.

Pusey [11] (1924) described the disease as follows:

The eruption may be general and abundant involving in some cases not only the skin but also the mucous membranes of the eyes and mouth . . . usually seen only in the severer forms of erythema multiforme. In the mouth they appear as rounded inflammatory erosions covered by a necrotic pellicle. In very rare cases, there is an acute febrile disturbance with temperature of 103-104 F. and severe pain in the joints resembling acute articular rheumatism. The constitutional disturbances . . . are likely to continue for some time . . . or until the eruption begins to subside . . . and many writers believe that it is an acute specific disease.

E. Fuchs [12] (1876) described a case of conjunctivitis in which pseudomembranes appeared on the palpebral conjunctiva of each eye. These membranes could be easily stripped off, but would rapidly re-form. The conjunctivitis was associated with a generalized eruption of the skin. The latter assumed the concentric vesicular type (herpes iris variety of erythema exudativum multiforme), whence he called the ocular condition herpes iris conjunctivae. The buccal mucous membranes showed lesions similar to those on the skin. There was a slight rise of temperature, and some constitutional disturbance. The patient left the hospital with the corneas normal. I am of the opinion, however, that the subsequent course of the illness would have revealed corneal complications.

Molènes-Mahon [13] (1884), in a monograph, stated:

One often observes it (polymorphous erythema) also as part of an infection not yet classified, with a group of decidedly distinct symptoms. For these cases, we propose the name infectious polymorphous erythema in the narrower sense.

Gerke and Kain [14] (1892), reported a case as follows:

Patient had a membranous deposit on the palpebral conjunctivae. In the mouth, upon the tongue and throat there was a number of whitish membranes. The temperature was elevated. A skin eruption appeared consisting of bright

10. Ormsby, O. S.: A Practical Treatise on Diseases of the Skin, ed. 2, Philadelphia, Lea & Febiger, 1921.

11. Pusey, W. A.: The Principles and Practice of Dermatology, New York, D. Appleton & Company, 1924.

12. Fuchs, E.: Klin. Monatsbl. f. Augenh. **14**:333, 1876.

13. de Molènes-Mahon, P.: Contribution à l'étude des maladies infectieuses, Paris, 1884.

14. Gerke and Kain: Ein Fall von Croup der Bindehaut, der Mund-und Rachenschleimhaut, Arch. f. Augenh. **24**:305, 1892.

red macules and papules involving mostly the extensor aspect of the hands and feet. Desquamation of skin eruption. Swelling of submaxillary glands.

These authors considered it a specific disease (morbus sui generis).

Dühring[15] (1896) gave the following detailed discussion of the disease:

In accordance with our experience which agrees with that of the native physicians, there appears in Turkey, a disease, epidemic in character, which proves to be an exanthematous infectious disease as shown by its symptoms . . . and which in its efflorescence corresponds to the disease described by Hebra as erythema exudativum multiforme. The lesions present much polymorphism as regards their shape and color, as well as their sequence. The principal basic forms we would designate the papular and vesicular.

Also, one meets with papules and vesicles upon the eyelids which may give rise to considerable swelling. In the majority of cases there is involvement of the conjunctivae and the mucous membranes that adjoin the face. Indeed, the quite characteristic conjunctivitis is frequently the first symptom. The disease appears in both the endemic and epidemic form. This contribution is based upon an epidemic comprising 105 cases. The buccal mucous membranes are affected in the majority of cases, the throat seldom. There are affected especially the mucous membrane of the upper and lower lips together with that of the adjoining cheeks, also, often the gums. The mucous membrane and skin lesions appear about the same time; in several cases, however, the pathology in the mucous membranes preceded the skin eruption. The mucous membrane lesions may be erythematous, papular, vesicular, or ulcerations may take place. The papular lesion is the most common one, and is especially distinct on the mucous membrane of the lips and resembles old hemorrhagic infiltrates. The color varies from reddish to bluish black. These lesions disappear simultaneously with the skin eruption, leaving no trace behind. The vesicular eruption on the mucous membranes partakes of the characteristics of this form of eruption on the skin. Ulcerations appear here very seldom, and then only when the skin eruption is very profuse and the general symptoms are pronounced. They are irregularly circumscribed, covered with yellow secretion, quite shallow losses of substance surrounded by narrow reddish areas of inflammation. Hardly, at all, or only casually is there mentioned in the literature a very important and constant manifestation of erythema exudativum multiforme (Hebra), namely, conjunctivitis exanthematica, which one may well designate conjunctivitis papulo-vesiculosa. Rigler, in 1852, speaks of conjunctivitis as frequently associated with erythema papulatum. The conjunctivitis is frequently the first objective manifestation of erythema exudativum multiforme. At times it appears a few days subsequent to the skin lesions. One first notes on the conjunctiva bulbi a variable number of circumscribed patches which are due to infiltration of the conjunctiva and of the underlying connective tissue. These patches vary in shape and extent. They are somewhat raised above the surface of the unaffected conjunctiva, and show small elevations which are due to circumscribed infiltrations of the subepithelium; they are yellowish or a light gray. The conjunctival and subconjunctival vessels are greatly injected, which gives to these parts a reddish appearance. The rest of the conjunctiva is hyperemic. The subjective symptoms are burning, itching, tearing, very mild photophobia, very little secretion. The disappearance of the conjunctivitis occurs simultaneously

15. Dühring, E.: Beitrag zur Lehre von den polymorphen Erythemen, Arch. f. Dermat. u. Syph. **35**:211, 1896.

with the skin eruption. Both eyes are affected. We have stated that this disease is an acute exanthematous infectious disease, and that it possesses all the earmarks of an infection, namely, incubation, invasion, eruption, termination of the eruption in a definite period, as well as the subjective and objective manifestations of a general eruptive disease. . . . The severity of the general symptoms, height of fever and the extent of the exanthem are in direct relationship to each other. Anemia is quite characteristic, even early in the disease. The lymph glands, especially the cervical, are enlarged. The spleen is enlarged to percussion.

Dühring's patients suffered, obviously, from only a mild type of conjunctivitis.

Victor Hanke[16] (1901) described two forms of herpes iris conjunctivae.

Herpes iris conjunctivae may appear in two forms, according to degree of severity, which forms can be differentiated clinically.

1. In the mildest form, it appears as a simple catarrhal affection. It differs from ordinary catarrhal conjunctivitis in that the subjective and objective manifestations are much less pronounced: the lids are not so swollen, the scleral conjunctiva is less injected, and there is absence of mucous or muco-purulent discharge, the course is more prolonged, and there are no sequelae. There is, however, a polymorphous skin eruption, and the various mucous surfaces may be involved.

2. The severe type of herpes iris conjunctivae partakes of the characteristics of a membranous conjunctivitis; and, in very severe cases the fornical and bulbar conjunctivae are involved, and there is present keratitis and corneal ulceration.

The case observed by us had the membrane extend upon the bulbar conjunctivae, and both corneae ulcerated (one superficially, the other continued on to perforation). On the entire body, were present numerous red spots, which were hemorrhagic in the center. At the same time the mucous membranes of the mouth and throat became affected in a similar manner, and apparently also the stomach and lungs. Soon thereafter, there appeared a diffuse membranous inflammation of the conjunctivae, and ulceration of the corneae due to keratitis suppurativa. The skin efflorescences turned into vesicles which ruptured and wounds resulted. Exitus lethalis occurred. The postmortem findings were: Erythema multiforme conjunctivarum, laryngis, pharyngis, et cutis, complicated by suppurative bronchopneumonia. Herpes iris (erythema exudativum multiforme) conjunctivae, assuming a membranous form and with sequelae, may occur as an isolated phenomenon, without at any time involvement of the skin or mouth and throat, just as erythema exudativum multiforme of the mouth and throat may occur without any other parts being implicated.

Hanke mentioned Mason's case, which was diagnosed as scarlatina. There was a membranous conjunctivitis and a similar inflammation in the oral cavity. The conjunctival pseudomembranes, which persisted for four months, spread over the corneas, causing necrosis of the latter. The end-result was bilateral corneal staphyloma. Hanke also quoted from the literature a number of cases of membranous conjunctivitis in which the corneas were affected, with marked visual impairment.

16. Hanke, Victor: Der Herpes iris des Auges, Arch. f. Ophth. **52**:263, 1901.

Hans Barkan [17] (1918) reported a case of membranous conjunctivitis with marked involvement of the mucous membranes of the mouth and lips and a bloody urethral discharge. The skin was covered with hemorrhagic blebs. A year previously, the patient had herpes iris of the skin and mucous membranes accompanied by a violent conjunctivitis. The lesions of the skin and mucous membrane healed, but the eyes remained unimproved. The patient eventually became practically blind, owing to corneal opacification. Many bands of adhesions traversed the conjunctival sac, and a profuse, stringy mucoid discharge persisted.

Stark's [18] patient (1918) was taken ill four years previously with a low grade fever lasting three weeks. There was a macular eruption resembling measles. Later, the eruption took on a bullous appearance. The mucous membranes of the mouth and throat showed many discrete membranes. The eyes were involved. The ocular sequelae were: persistent grayish membranes of the tarsal conjunctivae which quickly re-formed when removed, much scarring of the affected conjunctivae, several nebulae in the right cornea and vascularization and cloudiness of the left cornea.

Hartler [19] (1921) discussed two cases in which the patients had been admitted to the Copenhagen Contagious Disease Hospital for diphtheria. The tarsal conjunctivae were covered with a thick false membrane. The oral mucous membranes and lips showed many small discrete pseudomembranes. There was a generalized vesicular cutaneous eruption, which had dried and become crusted. The patients were acutely ill. About a month later, the corneas in the first patient commenced to reveal a faint turbidity, the final vision in both eyes being 20/60. At no time was the diphtheria bacillus, gonococcus or any other specific organism found. The second patient was discharged from the hospital without ocular complications. I believe, however, that if this case had been followed up corneal changes would have been noted.

Raffin [20] (1922) preferred to call the lesions aphthous rather than membranous. He stated that the primary eruption is aphthous, and that subsequently it is apt to become covered with coagulated fibrin, thus giving it a pseudomembranous character, or, as the case may be, converting it into ulcers. The patient was taken acutely ill with fever and an eruption of the mouth. A diagnosis of bronchial catarrh with

17. Barkan, Hans: Herpes Iris of the Conjunctiva, Arch. Ophth. **42**:236, 1913.

18. Stark, H. H.: Chronic Membraneous Conjunctivitis of Over Four Years' Duration, Am. J. Ophth. **1**:91, 1918.

19. Hartler, C. C.: Ueber die Kombination der Conjunctivitis et Stomatitis pseudomembranacea und ihr Verhaeltnis zum Erythema multiforme und Pemphigus, Klin. Monatsbl. f. Augenh. **67**:223, 1921.

20. Raffin, Albert: Conjunctivitis, Rhinitis und Stomatitis aphthosa mit Erythema multiforme, Klin. Monatsbl. f. Augenh. **68**:216, 1922.

aphthous stomatitis was made. The conjunctivae were inflamed and contained a pure culture of gram-positive diplococci. There was a generalized rash of the skin consisting of raised erythematous patches and vesicles. The mucous membranes of the lips, mouth and pharynx, as well as the conjunctivae were studded with discrete ulcers covered with yellowish disciform pseudomembranes. There was analgesia and anesthesia of both corneas. Pseudomembranes were also found in the nares. Streptococci were obtained in blood cultures. The leukocyte count varied from 9,000 to 16,000. The corneas became and remained diffusely cloudy. The conjunctivitis subsided, but scarring took place, followed by symblepharon. Scar formation also occurred in the nose, mouth and throat. Raffin called attention to the fact that some cases of foot and mouth diseases in man present an identical clinical picture.

Stevens and Johnson [1] (1922) described the cases of two children, who were seen ten and twenty-two days from the onset of the illness, They had high fever and a generalized eruption of the skin; the buccal mucosa was markedly inflamed, and there was a profuse purulent conjunctival discharge. The course of the disease was protracted. The patients recovered. In the first case, ulcers appeared in the left cornea with resultant scarring. In the second case, a bilateral suppurative keratitis with perforation and a purulent ophthalmia, ensued, the final result being phthisis bulbi and total blindness. Both cases were originally diagnosed as hemorrhagic measles. The authors stated:

> This syndrome suggests strongly an infectious disease of unknown etiology. We believe that this condition deserves to be considered a definite clinical entity.

Wheeler [21] (1930) reported the case of a child 8 years old, who was taken acutely ill with febrile symptoms. A generalized cutaneous rash appeared. Large grayish membranes developed in the mouth and throat. The eyes were inflamed, resulting in corneal ulceration. Later there was a profuse purulent conjunctival discharge. The corneal ulceration progressed and eventuated in perforation. The left eye became totally blind, the right had light perception only.

Rutherford [22] (1929) reported the case of a child aged 27 months, who had both eyes removed, the right because of corneal staphyloma and symblepharon, the left because of beginning atrophia bulbi due to ulceration of the cornea followed by perforation. This history is that the child became acutely ill, the condition having a protracted febrile course. The eyes, nose and throat were inflamed, and they presented a purulent discharge. On the tarsal surfaces of the conjunctivae, pseudo-

21. Wheeler, J. M.: Destructive Purulent Ophthalmia Accompanying an Eruptive Fever with Stomatitis, Am. J. Ophth. **13**:508, 1930.

22. Rutherford: Membraneous Conjunctivitis with Loss of Eyeballs, Tr. Sect. Ophth., A. M. A., 1929.

membranes formed which could be readily stripped off, but which would quickly re-form. The corneas soon became gravely affected. A maculopapular eruption of the skin developed. The lymph glands of the neck were enlarged. The leukocyte count was normal. Except for the bilateral anophthalmos, the patient recovered completely.

A careful examination of the literature from 1867 to the present, together with much thought given to the clinical features of this disease and their variations, convinces me that one is concerned not with a new clinical entity, but with a rare type of erythema exudativum multiforme (Hebra), unrecognized, as a rule, by ophthalmologist and dermatologist alike.

My patients were seen early in the course of the disease and were followed for more than two years, so that an opportunity was offered for observing and studying the evolution of the ocular picture as well as the sequelae and the permanent damage that resulted.

It is a disease that has a predilection for ectodermal structure; for, although the conjunctiva and cornea and the buccal mucosa bear the brunt of the attack, one recalls that these parts, commonly called mucous membranes, are simply modified skin and, like the latter, derived from the outer embryonic coat.

The distinguishing characteristics of the eruption of erythema exudativum multiforme (Hebra) are the sudden onset, rapid dissemination, preference for the extensor surfaces, symmetrical situation, polymorphism, long duration, unusual desquamation, paucity of discomfort, such as itching or burning, and the mucous membranes not infrequently affected. According to Unna and others, the histopathology of erythema exudativum multiforme is that of a serous infiltration of the epithelial and subepithelial layers accompanied by vascular dilatation and stasis. There is emigration of white cells and their aggregation about the subepithelial capillary network. When the inflammatory reaction is intense or the venous stasis marked, there is diapedesis of erythrocytes or even extravasation of blood, and thus petechiae or more extensive hemorrhagic areas develop. As the mucous surfaces are moist, do not have great resistance and are subjected to friction, especially in the case of the conjunctiva when a severe eruption appears on these parts it may break down and ulcers may occur; in the presence of much fibrinous exudate, these ulcers become covered with a coagulum, which results in the formation of a pseudomembrane. The severity of the rash may vary greatly both on the skin and on the mucous membranes. The conjunctival pathologic condition grades from simple hyperemia to a pseudomembranous process. The extent to which the skin is affected bears no relation to the involvement of the mucous membranes. The skin may be the seat of a florid eruption and the mucous membranes be free or slightly attacked, or the converse may obtain. Cases are reported

in which the disease was limited to the mucous surfaces of the mouth and throat, or even to the conjunctivae, a fact to be borne in mind in obscure conjunctivitides, especially of an aphthous or membranous type.

Many distinguished dermatologists aver that erythema exudativum multiforme (Hebra) is a systemic disease of infectious origin, and that the eruption is due to the action of toxins or to bacterial emboli. Although the offending organism has not been identified, several writers implicate some strain of the ubiquitous streptococcus residing in a focus such as the tonsils, teeth or gastro-intestinal tract. A recent investigator (Ramel,[23] 1930) ascribed the etiologic rôle to an attenuated Koch bacillus.

This disease should offer no difficulty from the standpoint of diagnosis, if its incidence only be borne in mind. It presents such a definite picture that once seen or described its recognition is surprisingly easy.

In the chronic stage, it may be mistaken for an old trachoma. In both instances, there are adhesions between the bulbar and palpebral conjunctivae, shallow or obliterated fornices and pannus. The history and the peculiar conjunctival discharge should remove all doubt.

Pemphigus vulgaris associated with conjunctival involvement may give rise to some confusion when the bullae that characterize this disease have ruptured and scarring has taken place. But the large vesicles, which become infected and crusted, the chronicity from the very incipiency of the condition, the absence of multiformity of the cutaneous manifestations and the invariably progressive and lethal course should eliminate any uncertainty. Parenthetically it may be stated that my first case was diagnosed by several eminent ophthalmologists as either pemphigus or trachoma; in fact, a bilateral trasectomy was performed in the belief that the tarsal plates were deformed as the result of a trachomatous process.

Acute pemphigus may simulate this disease. The conjunctiva and other mucous membranes may be covered by a false membrane, but the original lesion is bullous. In addition, there are all the symptoms of sepsis (chills, high fever, profound asthenia, etc.), for acute pemphigus is a true septicemia resulting from a trauma caused by the handling of dead animals. The disease is of short duration and terminates fatally in the majority of cases. The presence of acute pemphigus limited to the conjunctivae, is, at best, doubtful. One should not make a diagnosis of acute conjunctival pemphigus unless the characteristic cutaneous lesions are also present, namely, blebs in successive crops that rupture and become filled with pus or blood or form dirty crusts.

23. Ramel, Edwin: Some Observations on the Causation of Erythema Exudative Multiforme Hebra, Brit. J. Dermat. **42**:1 (Jan.) 1930.

It is true that pemphigus involving the conjunctiva is followed by symblepharon and xerosis conjunctivae, but these may occur in any violent inflammation of the conjunctiva when destruction of tissue ensues with resulting circatrization and occlusion of the ostia of the various glands that empty in the conjunctival sac. It is likely that many cases captioned acute pemphigus of the conjunctiva are actually instances of erythema exudativum multiforme (Hebra), in which the conjunctivae are seriously involved as part of a general disease process. This is still more likely, if the lesions of the skin are vesicular, or the brunt of the attack falls on the eyes. The so-called essential shrinking of the conjunctivae, which most authors consider to be a sequel of conjunctival pemphigus in the majority of cases, is doubtless the end-result of conjunctivitis membranacea vel aphthosa, and is a picture that finally obtained in my patients. The question arises whether erythema exudativum multiforme (Hebra), especially when it appears in the herpes iris form and implicates the conjunctivae, is not responsible for those cases of essential shrinking of the conjunctivae attributed to acute pemphigus.

In sepsis from various sources there may be a variety of cutaneous lesions. Circumscribed hemorrhages are among the most frequent; some are of considerable size, many only petechial. In septic sore throat with streptococcemia and cutaneous eruption, some confusion may arise in making the diagnosis. But here, the ocular structures exhibit only mild involvement or none at all, leukocytosis probably is present, a blood culture would likely be positive, and the eruption of the skin would be less varied and less selective as to site.

Agranulocytic angina, when accompanied by an eruption of the skin and conjunctivitis, may cause some difficulty in the differential diagnosis. The disease, however, is of grave prognostic significance, most of the cases terminating fatally. The angina assumes the appearance of necrotic ulcers, the lesions of the skin are not so diverse, the conjunctivitis is mild, and, of course, there is the pathognomonic blood picture—rapid diminution in the number of polymorphonuclear leukocytes and even their total extinction, so that the cells are 100 per cent lymphocytes, and progressive leukopenia, the white cells in many cases being less than 500 per cubic millimeter.

Vincent's angina with its distinctive ulceromembranous pharyngitis and stomatitis presents a picture in the mouth and throat identical with that obtained in my cases. But here the similarity ends, although my cases were diagnosed by the laryngologists as Vincent's angina. In that condition, however, there is no cutaneous eruption, the eyes are not affected, and the specific anaerobic micro-organism can be isolated.

It is interesting to note that when the disease has appeared in children it has usually been diagnosed as hemorrhagic measles. In fact, the

third patient in my series was admitted to a hospital for contagious diseases and treated for measles, the generalized petechial rash being responsible for the error. Measles does not reveal a multiplicity of lesions of the skin, the facies is characteristic, there is coryza and early involvement of the bronchial tree, the eyelids are tumid, the conjunctivitis is of the catarrhal type, and the course of the disease is quite different.

In those instances in which the rash assumes the form of unbilicated vesicles, it may remind one of the early stage of smallpox or varicella.

When limited to the conjunctiva or in association with pharyngeal diphtheria, diphtheria is easily differentiated by a culture and by the absence of a generalized polymorphous eruption of the skin.

Foot and mouth disease, a disease of cattle that is occasionally transmitted to human beings via milk or its products, when severe may furnish a strikingly similar picture. The conjunctiva may show aphthous patches with membrane formation. The lesions of the skin, however, are vesicular, and only when a wide area of the surface of the skin is involved, which is rare, does difficulty arise.

Pseudomembranous formation may be caused by any severe disease of the conjunctiva. With a copious exudate rich in fibrinous material, coagulation of the latter occurs, which produces a membrane that covers the conjunctival surface of the lid or of the bulb or both. Many nonspecific agencies may be the exciting factors (a variety of bacteria, including Klebs-Loeffler, gonococcus, streptococcus; chemical burns, e. g., lime, silver nitrate, acids, alkali; and physical traumas). The end-results of severe pseudomembranous conjunctivitides, whatever the causative factor, are marked impairment of vision and, objectively, symblepharon and xerosis conjunctivae. At this time, the differential diagnosis can be made only from the history of the case and a review of its clinical course.

When membranous or aphthous conjunctivitis is part of the clinical entity of erythema exudativum multiforme (Hebra), one must be guarded in one's prognostication as to the ultimate visual result. In these cases the sight is insidiously and progressively affected, in spite of the apparently hopeful outlook at the beginning. The impaired vision is due primarily to a corneal pathologic process, which consists of diffuse opacification and vascularization; or ulcers form with resulting dense opacities or with perforation followed by posterior synechiae or endophthalmia purulentosa with loss of the eyeball. The ocular pathologic process starts in the conjunctiva, and the cornea remains free for some time. After a variable period, the latter is secondarily invaded either as a result of the extension of the disease process or, more probably, through the medium of the excessively irritating conjunctival discharge.

SUMMARY

Erythema exudativum multiforme not infrequently implicates the eyes. The ocular pathologic process varies from hyperemia to destruction of tissue, in the latter instance often necessitating enucleation of the bulb. The corneal complications, which are responsible for the visual disaster, are delayed in their onset and insidiously progressive. In those cases in which, in spite of the virulence of the process, the eyeballs do not require removal, characteristic sequelae remain, namely, symblepharon, xerosis, a peculiar, stringy, viscid, conjunctival discharge and the aforementioned corneal lesions or the latter only. The disease is a distinct clinical entity and presents all the characteristics of an acute infectious disease. The caption, herpes iris conjunctivae, under which the disease was early described, is doubtless the cause for its remaining in obscurity so long and being ignored in the textbooks. Lately, I have been using large doses of bacterial vaccines intravenously with some promise of success.

ADVANCEMENTS AND OTHER SHORTENING OPERATIONS IN CONCOMITANT SQUINT*

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Twenty-one years of study and practice in the field of normal and pathologic ocular motility have led to certain impressions and convictions. These beliefs have taken form, first, as the result of the opinions of those who seem to me to have been the leaders in this special field of investigation, and, second, as the result of my personal study and experience. Certain rather definite conclusions, therefore, seem to be logical, and for the most part have received general acceptance. It is true that some of these conclusions are not accepted by all, but those who dissent have not been able to replace them by theories that will stand the test of minute analysis as well as the ones that I shall set forth as a working premise. As the surgical phases of squint are formulated on some of these generally accepted theories, it is important to state briefly a few of the facts that I believe have been established:

1. In the etiology of concomitant squint, a defective fusion faculty forms the background in the presence of which other factors, such as refractive errors and precipitating causes, are capable of bringing about the several forms of squint. The total absence of fusion leads to true alternating squint. A defective fusion faculty predisposes to a monolateral type.

2. In refractive errors, anisometropia is the dominating secondary influence which precipitates monocular squint, a defective fusion faculty being the primary etiologic factor.

3. In monocular esotropia, lowered central vision is due to amblyopia ex anopsia in all but a few instances, in which a small central scotoma. 3 degrees in diameter, can be demonstrated.

4. In this type of strabismus, the squint and the accompanying amblyopia can be transferred to the other eye in young children, and in all cases of acquired amblyopia central vision can be restored if training is practiced before the fifth or sixth year of life.

5. The fusion faculty, if present, can be awakened to normal activity in children under 7 years of age and, also, but to a less extent, in adults.

6. All treatment—refractive, orthoptic, fusional and surgical—accomplishes most good in children less than 6 years of age.

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Other important deductions might be made, but those mentioned have an important bearing on the surgical indications in concomitant squint.

In discussing the surgical treatment of squint, it is evident that each case becomes an individual study as to its surgical needs. An operator may have his preference for a special method, but the patient's interests are best served by a choice of the surgical procedure especially adapted to the individual case rather than the one preferred by the operator. In order to arrive at proper conclusions as to the patient's needs, in the first place, the case should be carefully studied and classified, and, in the second place, the value, limitations and indications of each surgical procedure should be properly appreciated.

In the first group of contingencies the character of the squint should be considered. Is it alternating or monolateral? If the latter, what is the state of the fusion faculty? Is amblyopia present and to what degree? Can the amblyopia be corrected, or is it beyond training? In either type, what is the angle of squint to be corrected? To simplify the analysis, can one hope to obtain single binocular vision, or can only cosmetic results be obtained? It is assumed that the refractive error has been properly corrected, that the fusion faculty has been developed to its maximum degree and that amblyopia ex anopsia has been corrected as far as possible at the time the operation becomes necessary. With increasing experience, I am advising operation as early as the third or fourth year, and some of this training follows rather than precedes the operation.

Given a careful analysis of each case as outlined, the choice of the operation becomes largely simplified if by actual experience one has determined the value of and indications for each type of operation.

Jameson [1] described the recession type of operation. I am asked to confine my discussion to advancements and other shortening methods which are practiced. With Dr. Jameson's permission, I may infringe slightly on his domain in a final summary.

From a popular standpoint, tendon-tucking is deserving of careful analysis. When properly performed, it is of distinct value, but is limited in its application. It is suitable in squint of moderate degree, i. e., a squint of 10 or 12 degrees, with maximum results in individual cases of 15 degrees. In fact, it is more applicable in the phorias. I am aware of the fact that some operators feel that it is suitable for squint of considerable degree. There are, however, certain underlying principles in operations on the muscles which must be thoroughly mastered and respected in order to obtain the ideal results. One of these principles is exactness and precision, which often are best obtained by dividing the work between two muscles, the internal and external rectus in the same

1. Jameson, P. C.: The Surgical Entity of Muscle Recession, Arch. Ophth., this issue p. 329.

eye, on one hand, and between the two external recti on the other hand. Which muscles should share in this division, in an individual case, can be determined only by the classification of the case as already given. A second principle, which is a corollary of the first, is the inadvisability of attempting too much by a single procedure. In the language of the golfer who attempts to reach the green with a mashie when a midiron is needed, pressing is apt to spoil the shot. If tucking is attempted, it should be used in a well selected case—one that falls easily within its range of possibilities. It is the easiest of all the legitimate operations that may be practiced in squint. One should not forget, however, that

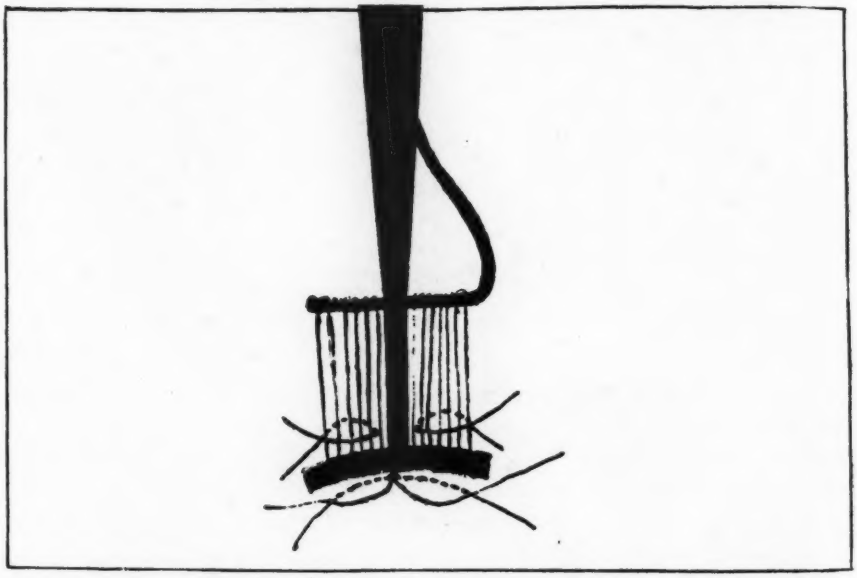

Diagram illustrating the positions of tucking sutures. The upper sutures are correctly placed. The lower sutures are apt to lead to necrosis of the tendon.

although the operation possesses advantages, it also has its disadvantages. From a purely cosmetic standpoint, the appearance of the field of operation after the healing is complete is not as satisfactory as that in an advancement or resection. The thickened area that remains is increased with attempts to correct wide deviations.

TECHNIC

The technic is worthy of analysis. Many methods have been suggested, as is shown by the great number of tucking instruments on exhibition by each instrument maker. Probably the best type is the Bishop tucker. It is light and compact, as ocular instruments should be. With it the sutures above and below the center of the shank can be well

and properly placed between the hook supporting the tendon and the foot of the tucker. When the sutures are so placed, the nutrition of the tendon is not disturbed because the center of the tendon is left intact. If this method is followed, there will be no occasion for necrosis of the knuckle of tendon (instances of which are reported in the literature), which undoubtedly is due to complete constriction of the tendon by improperly placed sutures.

The suture material is important. Some operators use no. 6 twisted silk. In some instances the sutures are buried. The method does not appeal to me, as there is no likelihood that complete absorption will occur, and the buried silk can only act as a foreign body and be a source of irritation. Twenty day chromicized catgut gives rise to tumefactions which eventually disappear, but an elevated area of conjunctiva is apt to remain. In my experience a 00 ten day chromic catgut is the most satisfactory suture material.

The vertical conjunctival wound is closed by a running suture of no. 1 twisted silk. Both eyes should be occluded for three days. The conjunctival suture may be removed on the fifth day, and on the sixth day the eye operated on may remain uncovered.

Although the surgical technic involved is simple from an operative standpoint, the operation gives rise to considerable trauma of the tissues. The capsule is much disturbed, and adhesions are more extensive than after a well performed resection or advancement when muscle, capsule and conjunctiva are included in the advancement forceps. Notwithstanding its general popularity, it does not have a strong appeal from the standpoint of refined surgery. When it is used, its limitations should be respected.

RESECTIONS AND ADVANCEMENTS

From the standpoint of good surgery, including sound surgical principles, maximum end-results and a satisfactory appearance of the field of operation after the healing process is complete, resections and advancements are the operations of choice in the group of procedures under discussion. Which of these procedures shall be selected in a given case is determined by the degree of squint to be corrected. The Worth operation will be discussed along with the Reese procedure. All probably agree that in simple resection of the muscle alone, the Reese operation is probably the best form of operation at one's disposal. In the group of procedures that shorten or advance the muscle, capsule and conjunctiva, the Worth operation is easily the leader.[2]

Those who operate for squint will agree that in convergent squint the conjunctiva and capsule over the external rectus, as well as the

2. It is the author's belief that the undersurface of the muscle should be lined by capsule as it is in the Worth operation.

muscle itself, are in a much relaxed and flabby condition. They lack in tonicity, and when the hook is placed beneath the muscle, this relaxed condition becomes very apparent. The capsule over the internal rectus and the muscle, on the contrary, are tense and rigid. The same conditions are observed in divergent squint, but are reversed: The internal rectus and capsule are thinned out and relaxed, and the external rectus and capsule are more rigid in contrast. This, in a large measures, accounts for the deficient rotations externally in convergent strabismus and defective internal rotation in divergent squint. There are other reasons for these defective rotations, but this is the chief one. In the application of surgical measures, therefore, the inclusion of the conjunctiva, capsule and muscle in the shortening process yields better corrective results than simple shortening of the muscle.

A second factor that commends the inclusion of capsule and conjunctiva is the greater respect for the tissues which the operation entails with fewer resulting adhesions and cicatricial bands. When the Worth operation is properly performed, the capsule is disturbed only to raise the muscle on the hook, the muscle being freed by a short incision above and below to admit of the application of the advancement forceps. After this is accomplished, the relation between the conjunctiva, capsule and muscle remains intact. There is less disturbance of the capsule in the Worth operation than in any other form of shortening, and, in the words of a well known general surgeon, "respect for tissues is one of the basic principles in good surgery."

A third important factor in any shortening operation on the external rectus is the ability of the operator to excise only a part of the tendon and to reattach the muscle by tendinous tissue rather than by the muscle itself. All muscles have tendinous insertions for obvious reasons. The tendon of the external rectus is 8.8 mm. in length, and if resection alone is not sufficient to correct the deviation, it is possible to increase the results by advancing the attachment in front of the tendon stump and yet continue to operate within the muscle tendon. Although much is said of reattachment of the advanced muscle to the cut tendon stump, regardless of the accuracy or inaccuracy of this observation, the fact remains that the shortening has been increased by reattachment in front of the stump. The gross gain, therefore, of advancement over a resection within what I believe are safe surgical limits for either operation is from 5 to 10 degrees of squint. A resection will yield the same results as a tucking operation. To this gain may be added for an advancement from 5 to 10 degrees more.

Three objections are raised against advancements: (1) danger of torsion of the vertical corneal meridian by reattachment too high or too low; (2) danger of perforation of the sclera by insertion of the needle

too deeply, and (3) danger of slipping or cutting out of the sutures by too superficial a bite in the episcleral tissues. The three objections are predicated on poor surgical technic and can not be charged against the operation.

If the guide suture is properly placed in the Worth operation and the two main sutures are equally well placed in relation to the guide suture, torsion and hyperphoria need not be feared. The same unfair objection could be raised against a poorly performed Reese resection or a badly executed tucking.

The second and third objections are equally the result of poor technic. The episcleral tissue in front of the stump should be carefully cleared, a needle with a good point should be carefully followed through the episcleral tissue, and neither the second nor the third accident should be encountered. In addition to poor surgical technic, other factors may be responsible for the last-mentioned accident. One of these factors is the mistake of attempting to overcome too much squint by a single operation. It involves the breaking of a well known surgical principle not to place too much tension on sutures. This can be overcome by attempting less or by dividing the work between two muscles: a double advancement or a recession of the opposing internal rectus. A second factor which should not be ignored is the bandaging of both eyes for four or five days, or until the healing process is under way.

Like the modern operation for cataract, the modern operation on the muscles calls for more refined technic than the simpler procedures of the past. In monocular squint, one no longer hopes for cosmetic results; single binocular vision is the goal. The procedures therefore call for greater precision and accuracy, and more thought must be given to each case to obtain the ideal results. It is not a question of what operation offers the least surgical difficulty, but of what procedure will adequately and accurately yield the results that one sets out to accomplish. No single operation will answer all of the modern requirements.

An operation that may be included in the group that I am discussing is the O'Connor cinch method of shortening. I hesitate to discuss the method until I have more thoroughly familiarized myself with what it may accomplish. Certain advantages are claimed for it by the author; namely, one need not bandage the eye not operated on, convalescence is rapid and the amount of shortening can be controlled by withdrawing the dermal strands from time to time until a perfect correction is secured. To offset these advantages, Irvine of Los Angeles stated that he abandoned the operation because of the annoying tumefactions that occur as in the tucking operation. Barkan of San Francisco leaves the strands in position for three weeks.

O'Connor in a recent letter stated that he finds the surgical correction of the phorias much more satisfactory than that of squint. As he practices the cinch operation in both groups of cases, I assume that the cinch operation does not yield the ideal results in squint that one may obtain by other methods. Although, in my experience, hyperphoria can be accurately corrected by other methods as well as by O'Connor's technic, the lateral phorias are not as easily corrected by surgical means as is monocular esotropia. From a theoretical standpoint, it does not appeal to me because of the underlying principles and the trauma to the tissues which it involves. I hope to be in a position to discuss the method after greater experience with the operation.

To summarize the surgical indications for the several operations which seem to be of value in modern operations on the muscles, I shall touch briefly on the field that Jameson [1] has covered. In order to designate succinctly what the procedures should be in a given case, the underlying principles and the advantages of recessions and shortening operations should briefly be compared. Both types of operation are necessary to meet the demands of clinical needs. Advancements or shortening operations restore tonicity and increase the function of subnormally acting muscles. Recessions reduce the activity of overacting muscles. In the main, restoring underacting muscles to normal function may be considered as a basically sound surgical principle. It also increases the power of the obliques, the two synergists of the external rectus, especially in their external rotating power and in torsion. Breaking the power of the internal rectus tends toward the same results, but not to the same extent after the squint is thoroughly established. Furthermore, in attempting to restore single binocular vision, the question of sustaining a normal convergence for the near-point must be considered. If the eyes, therefore, can be straightened by a constructive operation on the external recti, the future comfort of the patient in near work is more effectually assured. In evaluating the merits and advantages of the two types of operations, it is generally believed that shortening procedures should be given the preference and that recessions should be held in reserve as supplementary operations. As the final summary will indicate, however, there are instances in which a recession instead of a second shortening procedure may be selected.

CONCLUSIONS

From personal experience and from a digest of the literature, the following conclusions seem to be justified:

1. Tenotomies should not be practiced either in convergent or in divergent squint. Complete tenotomies are dangerous and partial tenotomies are of little help.

2. When a recession operation is indicated, the muscle should be so placed and reattached to the sclera that convergence is not reduced beyond what is required by the patient in sustained work at the near-point.

3. Advancement operations, including the several types discussed, are logical and constructive surgical procedures in all cases in which they are sufficient to correct the squint.

4. When squint of considerable degree is to be corrected, the logical procedure is an advancement, supplemented by a recession of the opposing muscle.

When more than one muscle must be operated on the choice between an advancement and a recession on the same eye or double advancements divided between the two eyes is largely determined: (1) by the character of the squint (whether it is alternating or monolateral), (2) by the presence or absence of incurable amblyopia and (3) by the exercise of good surgical judgment. A fourth factor should be considered in operations under local anesthesia: Any operation on the internal rectus is much more painful and tedious than the same operation on the external rectus. This may be a factor worth considering.

If the squint is alternating, as convergence is inconsequential the operation should be confined to one eye as far as possible. If the amblyopia is of long standing and cannot be corrected, the case logically falls into the same category, and the operation may be limited to the squinting eye.

If the squint is monocular and amblyopia can be corrected, it becomes a question of good surgical judgment as to whether the operation shall be performed on the two eyes, or be limited to one eye. My preference is for the former, but much may be said in favor of limiting the procedures to the squinting eye. An advancement and a recession on the same eye can be performed at one sitting. Double advancements are practiced by the discriminating surgeon, but not at one session, the second being performed about one or two weeks after the first. If convergence can be preserved, many surgeons will elect to perform an advancement and a recession at the same time, in order to avoid two operative sessions.

Finally, in order to furnish the younger ophthalmologists with a somewhat definite guide, I cite the following procedures that guide me in my operations on muscles although this classification must necessarily be regarded as elastic: (*a*) a squint of 10 degrees, resection or tucking; (*b*) a squint of 15 degrees, resection with advancement if necessary; (*c*) a squint of 20 degrees, resection with advancement (supplemented by a recession if necessary); (*d*) a squint of from 25 to 30 degrees.

(1) double advancements; (2) advancement and recession on one eye, and, if necessary, an advancement on the other side; (*e*) a squint of 30 or more degrees, double advancements with one or double recessions, if necessary, at two sittings, from seven to fourteen days apart; (*f*) in types *c* and *d*, a choice of double advancements, or an advancement and a recession. In divergent squint, the same rules apply, with this exception: Any surgical procedure is from 30 to 40 per cent more effective in convergent than in divergent squint. Although the underlying surgical principles are the same in each type, varying degrees of divergence require slight readjustments of supplementary operations to yield the best results. Furthermore, the prospects of obtaining single binocular vision in monocular exotropia are not as good as in monocular esotropia.

OCULAR CONVERGENCE: ITS RELATION TO BASAL METABOLISM AND ENDOCRINE DISEASE

A PRELIMINARY REPORT *

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In 1891, Möbius [1] pointed out that unilateral convergence is normal in exophthalmic goiter, but that in spite of muscular palsy or mechanical interference due to extreme protrusion of the eyeballs the act of binocular convergence is diminished.

In all modern textbooks relating to general medicine, the endocrine glands, ophthalmology or neurology, mention is made of the so-called Möbius sign of insufficiency of convergence in exophthalmic goiter. Osler,[2] Anders [3] and Blumer [4] had little to add except to call attention to the sign, and the words of Price,[5] "Various symptoms have been associated with exophthalmos, and although they do not add materially to the ease or certainty of diagnosis they are traditions of medicine," are suggestive of the attitude taken concerning the significance of insufficiency of convergence in general diseases.

Sahli,[6] in his textbook on diagnostic methods, drew the following conclusion:

> In spite of such subjective and objective confirmation of insufficiency of convergence, the degree of convergence which is transitorily accomplished by a vigorous voluntary impulse is in such cases often times considerable, so that the Landolt ophthalmodynamometer (which measures the convergence attainable by the maximum voluntary effort) does not suffice to demonstrate the insufficiency in exophthalmic goitre.

From an intensive search of the available literature no specific incident can be cited in which there was an attempt to correlate insufficiency of convergence and the basal metabolism. Holloway, Fry and

* Submitted for publication, Feb. 16, 1931.

1. Möbius: Deutsche Ztschr. f. Nervenh. **1**:189, 1891.
2. Osler: Principles and Practice of Medicine, New York, D. Appleton & Company.
3. Anders: Practice of Medicine, Philadelphia, W. B. Saunders Company, 1915.
4. Blumer: Bedside Diagnosis, Philadelphia, W. B. Saunders Company, 1928.
5. Price: A Textbook of the Practice of Medicine, ed. 3, London, Oxford University Press, 1929.
6. Sahli: Treatise on Diagnostic Methods of Examination, ed. 2, Philadelphia, W. B. Saunders Company.

Wentworth,[7] in a discourse on ocular signs in goiter, although they did not correlate insufficiency of convergence and the basal metabolism, nevertheless gave the following figures concerning the frequency of Möbius' sign: in hyperplastic toxic goiter, 10.2 per cent; in toxic adenoma, 40.9 per cent, and in nontoxic conditions, 14.3 per cent. These figures represent the percentage of cases that showed a convergence near point greater than 9 cm., approximately 3½ inches, from the base of the nose. In their entire series of 100 cases, a convergence near point greater than 9 cm. was found in only 19 per cent.

In routine examinations including tests of muscular function, ophthalmologists are continuously confronted with cases of weakness of

Results of Tests in One Hundred and Fifty Cases

No.	Type of Case	Average Convergence Near Point	Average Basal Metabolism	Average of Plus Rates	Average of Minus Rates
150	All	3.9	+ 4.9	23.1	9.9
64	All diagnosed	3.4+	+11.3	28.5	9.3
86	Not diagnosed	3.2+	— 1.0	16.5	13.7
14	Toxic adenoma	4.4+	+34.6	42.1	10.1
13	Obesity	2.6+	— 1.5	11.7	7.6
12	Hyperthyroidism	3.6+	+26.0	26.0	0.0
11	Hypothyroidism	2.1+	— 9.0	0.0	9.0
5	After thyroidectomy	2.9+	+ 2.4	28.0	14.0
4	Hypofunction of other endocrine glands	2.5+	3.2	9.0	7.3
3	Myxedema	4.0+	13.3	0.0	13.3
3	Tuberculosis	3.8+	+ 3.0	11.0	13.0
3	Tachycardia	4.6+	+18.0	18.0	0.0
2	Diabetes	3.0	+24.0	24.0	0.0
2	Surgical menopause	4.0	— 0.5	0.0	1.0
2	Asthma and bronchitis	5.0	+ 8.5	16.0	1.0
1	Sarcoma of skull	2.5	—14.0	0.0	14.0
1	Purpura	2.5	—10.0	0.0	10.0

convergence, and each operator has an individual point of view as to the importance of this phenomenon from both the ocular standpoint and that of general health. Is convergence insufficiency a prodromal sign of impending exophthalmic goiter? Is it only an accompaniment of the disease? Has it, perhaps, only a hereditary connection? Has it no relation to exophthalmic goiter? Is it of importance? These and many others are the questions that come to one's mind. Some think so little of the muscular response that tests of muscular balance are entirely eliminated from routine examinations. However, it is definite that there are persons who are greatly inconvenienced by inability to hold convergence for a reasonable length of time. Prisms or prism exercises have been used for this condition; both have their exponents. Operation on the offending muscle is often undertaken, but the general metabolism of the patient is often disregarded. Has this a definite

7. Holloway, T. B.; Fry, W. E., and Wentworth, H. A.: Ocular Signs in 100 Unselected Cases of Goiter, J. A. M. A. **92**:35 (Jan. 5) 1929.

bearing on the matter? With these ideas in mind, it was thought that a comparison of the basal metabolism and ability to converge might throw some light on this aspect of the problem.

The following account is a record of patients who presented themselves for tests of basal metabolism at the Jewish Hospital of St. Louis over a period of eight months, from March, 1928, to October, 1928, inclusive. The cases were recorded in chronological order; some of the patients were outpatients from the clinic or from private practice, and others, the usual run of hospitalized patients. The attending physician's diagnosis, if made, and doubtful or preliminary diagnoses are recorded. Cases without a definite diagnosis are also listed. In this series 150 patients were examined. Ocular convergence or the ability to converge was measured in inches as that point distant from the zygoma at which the patient was no longer able to hold the eyes in while viewing an approaching object. A pencil point was the object of choice. Few normal persons were found owing to the fact that a metabolism test had been thought necessary because of dysfunction.

Conclusive evidence is not obtainable from such a small series of cases, and it is my hope that this problem will be studied further by those interested. Kessel, Lieb and Hyman,[8] and later Kessel and Hyman,[9] working alone on studies of exophthalmic goiter and the involuntary nervous system, placed little if any importance on Möbius' sign. The accompanying table shows the number of cases in each group of typed conditions, the average for the convergence near point of each group, the average basal metabolism of each group, and also the averages of the plus and minus results.

COMMENT

It is interesting to note that the average convergence near point in all cases was 3.9 inches (9.9 cm.), which is slightly higher than the arbitrary figure taken by Holloway and his co-workers as normal, while at the same time the average basal metabolism was + 4.9 above the normal. The cases of toxic adenoma together with those of tachycardia showed poor convergence together with high basal metabolic rates. The cases of obesity and hypothyroidism showed both a low basal metabolism and a satisfactory ability to converge. Why the cases of myxedema in which the basal metabolic rate was expectedly in the minus column showed such a weakness of convergence cannot be satisfactorily explained.

104 South Michigan Avenue.

8. Kessel; Lieb, and Hyman: Studies of Graves' Syndrome and the Involuntary Nervous System, Am. J. M. Sc. **165**:384, 1923.

9. Kessel and Hyman: Am. J. M. Sc. **165**:513, 1923.

TUBERCULOUS KERATITIS

DESCEMETOCELE, PERFORATION, REPAIR; REPORT OF A CASE *

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The clinical course of cases of tuberculous keratitis has been reported so frequently that the progress in these cases, the improvement, the relapses, the treatment and the results have become familiar subjects of discussion and of papers in which the details of exhaustive studies have been given. In the brief presentation of this case I purpose to show three unusual features of the disease: (1) the destructive process deep in the corneal substance, (2) the formation of a descemetocele with subsequent perforation, and (3) the repair of the defect and apparent arrest of the process.

REPORT OF CASE

History.—A woman, aged 21, a hairdresser, apparently well nourished and in good health, came to the New York Eye and Ear Infirmary in December, 1926, complaining of recurring attacks of inflammation and pain in the left eye which had occurred almost continuously over a period of about five years. She had received treatment during this time, but she repeatedly suffered from a relapse or a recurrence of the disease, until at the time of examination the vision had been greatly reduced, and this together with increasing "scar" formation in the cornea had led her to seek further advice.

The family history disclosed that her father had died of tuberculosis four years previously, or about one year after the date on which the patient had experienced the first attack of the ocular disease. It was during his illness that her eye had become involved. Her mother was living and well. Two brothers were well, but one of them had an involvement of the lungs believed to be pneumonia. One sister was living, but was not well; she was delicate and subject to colds. Two sisters had died early in childhood of unknown causes.

The ocular history of the family was not important in relation to the patient's ocular complaint. Except for chickenpox, measles and whooping cough during childhood, the previous medical history was irrelevant. The patient had always been well, and her eyes had been normal in every respect, except for an inflammation of the right eye about one and one-half years before the left eye became involved. The cause of the inflammation of the right eye had not been determined by the oculist, but it was relieved after several weeks of treatment. Since that time the right eye had been strong and apparently normal.

Examination.—On examination, the right eye was found to be normal. The left eye was moderately inflamed, with mild deep circumcorneal injection, and

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* Read before the New York Academy of Medicine, Section of Ophthalmology, March 16, 1931.

the cornea was involved by an infiltration of the more superficial layer of the stroma, together with three more or less confluent, densely opaque areas in the deeper layers of the substance. These areas were centrally located; one of them extended nasalward over the margin of the iris and was more deeply infiltrated than the other portions of the lesion. These irregularly roundish areas were not typically papular, but were rather stippled or granular, similar to those in certain febrile types of keratitis. The superficial cornea was also affected; the epithelium was broken and lay irregularly in and about the lesions. The iris was moderately congested, and the pupil was slightly sluggish in reaction. The vision of the right eye was 20/16; that of the left eye, 20/200.

The patient was admitted to the infirmary for a diagnosis of the condition, and the routine laboratory examinations gave no definite information. Urinalysis and complete examinations of the blood, including the Wassermann test, gave negative results. Rhinologic examination and roentgenograms of the sinuses showed no indication of a pathologic process. A roentgenogram of the teeth was negative. The reaction to 1 mg. of old tuberculin was positive locally; the temperature reached 101 F., and the eye responded by a definite improvement in the reaction and by relief from pain in the course of forty-eight hours, from which the patient insisted she had rarely been free for at least a month previously. Dr. Imboden reported on the roentgenograms of the lungs as follows: "The thorax shows no indication of disease of the lungs. There is no evidence of a mediastinal disturbance. The diaphragm moves freely on respiration."

Treatment and Course.—I prescribed the regular serial treatment with tuberculin, a suitable diet, regular habits of sleep and rest and an open-air and sun-exposed environment. The cornea responded to this routine therapy by healing rapidly; the reaction subsided, and a thin nebulous scar formed in the superficial stroma, but over the deeply involved nasal portion there was some loss of substance with a throwing off of débris until only Descemet's membrane remained. A small cavity developed in the cornea (tuberculous?); the limiting walls were formed by fine lines of fibrous tissue, and were smooth and sharply round. The contents of the cavity had apparently undergone liquefaction, and thus had been thrown off into the conjunctival flora. Later a hernia of Descemet's membrane (descemetocele), about the size of a pinhead, gradually formed, and protruded slightly above the level of the surface of the cornea. This condition developed slowly over a month, the eye being entirely quiet and without pain. The vision improved slightly to 20/50, and there was a gain of 10 pounds (4.5 Kg.). The patient returned to work two months afterward. The routine care and the treatment with tuberculin, with a gradual increase in the interval between injections, were faithfully continued for about two years, without a relapse of the corneal process.

On May 12, 1929, three and one-half years later, the patient believed that she struck the eye against the branch of a tree; it felt as though a branch had penetrated the eye. She was not sure that this was true, but such was her impression from the incident and the severe pain she experienced at the time. On examination, the descemetocele was found to have been torn; in fact, under the slit-lamp only a small remnant of Descemet's membrane could be detected along the posterior margin of what was a sharply limited, perfectly round hole in the cornea, measuring 2 mm. in diameter. The corneal substance about the perforation apparently was not affected by the relapse of the tuberculous process, though this could not be definitely determined because of the condition of the eye. The pupillary margin of the iris partly filled the perforation; the anterior chamber had collapsed, and the usual conjunctival and iritic reaction was present.

The patient was sent to the infirmary at once, where local treatment was carried out, a bandage was applied and she was confined to bed. The anterior chamber remained collapsed for two weeks, in spite of repeated attempts to induce closure of the perforation. Pilocarpine was instilled in order to carry the iris more completely into the perforation, but the margins were too sharply limited and rounded, the result of a chronic process, and were therefore without an activating substance. As this procedure failed and cauterization of the area did not yield a result, the eye became more violently injected and painful, and because of this and the danger of subsequent involvement of the lens, a large flap of conjunctiva was dissected up and sutured over the perforation. After forty-eight hours, the flap failed to hold its position, as an attachment to the corneal lips of the wound did not take place, and the hole in the cornea was as patent as before.

Another attempt was made to close the hole by performing an iridectomy of the pupillary margin through the perforation, scarifying thoroughly the corneal margins of the hole and then bringing the entire pericorneal conjunctiva over the cornea by means of two large flaps, one from above and one from below; the flaps were tightly held in position by three mattress sutures. These conjunctival flaps were permitted to remain thus for two weeks, when the temporal mattress suture was released; one week later, the central suture was severed. The anterior chamber had been restored, the iris was free, and the nasal portion of the conjunctiva was firmly attached over the perforation. This attached portion was permitted to remain in position for four months; a firm dressing was applied for the purpose of supporting the flap, in order to secure a thoroughly healed wound and to supply sufficient local nourishment through the conjunctival blood vessels.

Five months after operation the conjunctival attachment was dissected free from the cornea. A small but slightly elevated scar remained, as the drawing of the eye illustrates (fig. 1). It was noted that five blood vessels passed from the nasal limbus through the substance of the cornea to the corneal lesion. The corneal scar about it seemed less dense and opaque than before; the iris was normal, and there was no inflammatory reaction. This condition of the eye has been maintained for almost two years.

COMMENT

The bacteriologic diagnosis could not be established in this case, because the cultures and smears from the lesion showed only cellular débris and a few cocci. However, the family history of tuberculosis (the corneal disease occurring about the time of the father's illness and death from tuberculosis), the positive response to the inoculation of tuberculin, the prompt resolution of the corneal process following routine care and treatment with tuberculin, and the subacute and chronic relapsing character of the corneal lesion seem to justify a fairly definite clinical diagnosis of tuberculous keratitis. The clinical changes in the lesion during the period of about ten years of involvement seem worthy of note, especially because during the process of resolution the deeper portion of the lesion evidently underwent a fibrous walling-off of the infection, with subsequent liquefaction of its cellular contents, which escaped in the conjunctival flora, and with formation of a small cavity in the cornea typical of tuberculous processes in other

Fig. 1.—Conjunctival transplant covering the perforation, two years after operation.

structures of the body. These pathologic changes, although observed only clinically, are thus interpreted as the result of frequent examinations under the slit-lamp microscope.

Aside from these pathologic changes, the clinical course of this case is of interest. The first stage of the disease, that of subacute or acutely relapsing attacks, extended over a period of five years previous to my observations. Each attack and each recession of the active process during that time produced the lesion, in part deep cellular infiltrate and in part

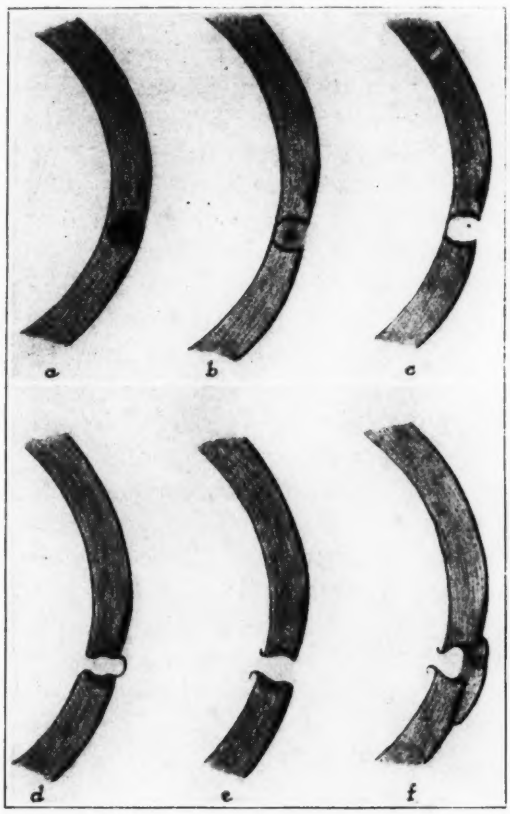

Fig. 2.—Diagrammatic horizontal sections at different stages of the corneal process: *a*, infiltration after five years; *b*, liquefaction, over a period of from four to six weeks; *c*, cavity, a period of two weeks; *d*, descemetocele, a period of three and one-half years; *e*, perforation, a period of from three to four weeks; *f*, conjunctival covering, a period of two years.

fibrous tissue change (fig. 2 *a*). The second stage was that noted after treatment with tuberculin and other routine care. In this period of resolution and repair, from about four to six weeks, the deeply infiltrated area appeared to soften (liquefy) and partly empty itself (fig. 2 *b*), and in the course of another few days it remained as a small

cavity in the cornea (fig. 2 *c*). About this time the patient was discharged from the hospital, and at the end of another week a hernia of Descemet's membrane had occurred (fig. 2 *d*). This stage or period of the descemetocele lasted for over three years without apparent change until injury occurred, when in reality there may have been a spontaneous rupture of Descemet's membrane as the patient with some uncertainty attributed the lesion to the apparent striking of the eye. The perforation or fistula of the cornea persisted for about three or four weeks (fig. 2 *e*), when it was calculated that perhaps the large conjunctival flap may have closed it, and the anterior chamber had been restored. Then followed the period of five months during which the conjunctival flap was permitted to remain attached to the perforation. This step was perhaps unnecessarily prolonged, but for obvious reasons was believed to be justified. Almost two years have passed since the conjunctival flap was dissected from its attachment (fig. 2 *f*), and while during this period the eye has been free from disease or any other change except an attack of catarrhal conjunctivitis, extreme caution has been used in protecting and favoring the eye in every way. Injections of tuberculin have been given at intervals of one month.

After almost ten years of involvement, as related, the eye is entirely quiet; there is no discomfort, but at times a consciousness of the smooth elevated thickening at the site of perforation. This area is not densely fibrous, but appears to be of the consistency of subconjunctival tissue, as though soft and slightly edematous, perhaps with a tiny fistulation (fig. 2 *f*). Although this is true and perforation may eventually occur, no other evidence that this may happen has been present.

To what degree the conjunctival flap may have contributed to the nourishment of the cornea in the control of the tuberculous process seems to be mere speculation in this case, because the corneal disease had been under control three and one-half years before the conjunctival flap was employed to close the perforation. Nevertheless, the deep and apparently permanent blood vessels passing through corneal substance to the lesion suggest a means of nourishment and support for corneal lesions of this character. This is especially true when one considers what may have been the result in this case had the conjunctival flap been used at the time of definite resolution of the corneal lesion before a descemetocele developed or even after the descemetocele developed but before perforation occurred.

Cosmetically, the eye is not all that one would like it to be, but its present appearance obviously is the result of the necessity for preservation of the globe. The possibility of making an Elschnig trephine transplantation of corneal substance has been considered, but the history of the case, the pathologic changes and the absence of Descemet's membrane

have not encouraged such a procedure. This, however, may have been justified before perforation occurred, and I believe that it should be considered in cases of descemetocele in which one may anticipate eventual perforation. It is well known that fistula of the cornea is commonly cured by support from a conjunctival flap, but this case presents features that are new in my experience — the size of the opening (large, round and fibrous), the chronic character of the perforation and the pathologic cause. Should perforation recur, the difficulty of again using the conjunctival flap as a support is evident, since friability of the conjunctiva has been increased incident to the previous operation and the formation of fibrous subconjunctival changes. If it should be impossible to use the conjunctival flap, would one be justified in attempting the Elschnig trephine corneal transplantation at the site of the perforation?

AN UNUSUAL CORNEAL OPACITY

REPORT OF A CASE *

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This case of opacity of the cornea is reported because of its rarity and its obscure nature and origin. The opacity was confined to the anterior layers of the substantia propria. It had the appearance of a "snow-storm" when looked at with focal illumination, and consisted of grayish-white round dots under the slit-lamp. Stereographs and a colored drawing illustrate this unusual corneal condition.

REPORT OF CASE

J. H., a Russian Jew, aged 38, first presented himself for examination on March 10, 1930, at the Eye Clinic of the Union Health Center. He complained of a diminution of vision in the right eye and lacrimation of both eyes while at work. He stated that he had good vision in both eyes until six years before, when one of his co-workers "playing a trick on him" forced some paint or shellac into his right eye. The eye became red and painful, and he was forced to go to an eye clinic, where he was treated for ten days. From that time the vision of this eye was foggy. His past history was irrelevant, and the only history of abnormality in his family was that of his child, aged 3, who had been wearing a cast on the left thigh for eighteen months. All members of his family had good vision as far as he knew.

On examination the lids, conjunctiva and lacrimal passages showed no abnormality, and the muscle balance was normal. The pupil was about 3 mm. in diameter, round and reacted well to light and in accommodation. The anterior chamber was of normal depth, the iris was brown and there were no posterior synechiae or vitreous opacities. The fundus was seen well and appeared normal, notwithstanding the corneal opacity. There was no change in the surface and luster of the cornea. Focal illumination with the Zeiss loupe showed numerous fine, round, grayish opacities, varying in size from that of a pinpoint to that of a pinhead. The opacities were deeply situated and diffusely spread over the cornea, leaving in the periphery a clear zone from 2 to 3 mm. in width. These dots were distributed at about an equal distance from each other, being more numerous in the center of the cornea, which had the appearance of being loosely tattooed with fine sand. The sensitivity of the cornea was not diminished.

The observations made with the slit-lamp were interesting. The epithelium and Bowman's membrane were clear. No superficial or deep blood vessels were present. The opacities were located in the anterior layers of the substantia propria of the cornea. No opacities could be seen in the deepest layers. The dots showed no tendency to confluence and were separated from each other by clear areas. The color of the dots was grayish white. In the center a few could be seen which had a brownish color. They all appeared refractile. The deep layers and the

* Submitted for publication, March 17, 1931.

endothelium seemed to be free from disease. There was no pigmentation or precipitates visible on the posterior surface of the cornea, and no pupillary membrane was present. The tension and visual fields were normal. The vision of this eye was 20/70, improved to 20/50 with a +0.50 sphere.

The left eye was normal. Examination by slit-lamp did not reveal any deposits or pigmentation in the perfectly transparent cornea. The vision of the left eye was 20/20 (emmetropia).

The Wassermann reaction was negative; the urine was normal; the teeth and tonsils showed no pathologic process. A routine analysis of the blood gave results

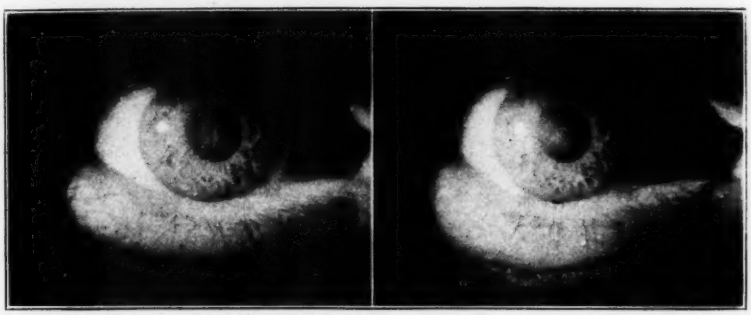

Fig. 1.—A stereoscopic photograph under medium power, taken by Dr. W. L. Hughes.

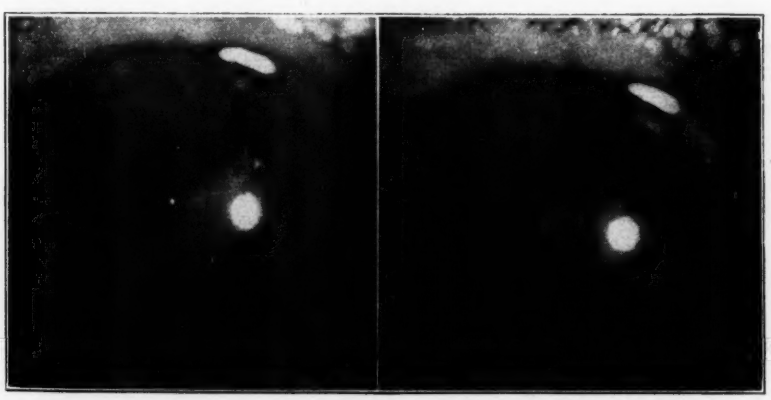

Fig. 2.—A stereoscopic photograph under high magnification, taken by Dr. W. L. Hughes.

well within normal limits: nonprotein nitrogen, 26.20 mg. per hundred cubic centimeters; urea, 13.10 mg.; uric acid, 2.30 mg.; blood sugar, 82.00 mg.; blood cholesterol, 0.149 mg., and calcium, 9.80 mg. A general physical examination gave negative results.

Because of the possible history of tuberculosis of the joint in one member of the patient's family, I was particularly careful with the tuberculin test. A Mantoux test was performed twice. In the first test, 0.1 cc. of a 1:100,000 dilution of old tuberculin was introduced. In ten days the second test was done, a 1:10,000 and a 1:1,000 dilution being used. Both tests gave a negative result, with no local or systemic reaction.

COMMENT

In discussing the nature and etiology of this corneal condition, one should consider whether its origin is dystrophic, systemic or congenital. Dystrophies of the cornea are usually bilateral, noninflammatory, degenerative processes which develop very slowly and which are characterized by deposits of hyaline bodies, cholesterol crystals, lime salts and other substances in the previously sound cornea. According to Axenfeld,[1] calcareous, amyloid, hyaline and fatty degeneration is the deposition of white, or yellowish-white granules in corneal scars, or in eyes in which nutrition is impaired owing to various causes. He expressed the belief that the deposit of these granules is possible because the cement substance of the corneal lamellae is diseased. According to Fuchs,[2] zonular opacities occur during and following severe intra-ocular diseases. They are a frequent type of dystrophy which forms in the plane of the palpebral fissure, "where, owing to evaporation, the lymph becomes more inspissated, so that the compound of lime precipitates." The opacity is composed of minute white or gray dots of lime and lies either in the epithelium or in Bowman's membrane, these tissues showing more predilection for lime salts than the other layers of the cornea. Groenouw's [3] nodular opacity of the cornea or grill-like keratitis is bilateral and progressive over a period of years. The lesion is in the superficial layers of the corneal stroma, causing bulging of the epithelium; anesthesia of the cornea is present, due to some neurotrophic disturbance.

In 1925, Gifford [4] described three cases of corneal dystrophy somewhat similar to mine. In his cases, however, edema and vacuoles were present in the epithelium, and elevations resembling fine droplets were seen on the endothelium. The opacities in my case involved the anterior layers of the substantia propria and did not bear any resemblance to the aforementioned dystrophies.

Corneal conditions due to systemic diseases may present changes somewhat similar to those observed in the cornea of my patient. Spanlang [5] reported a case of bilateral disease of the cornea in which irregular, whitish-yellow opacities involved the entire corneal stroma, leaving a transparent narrow rim of cornea at the periphery. A piece of the

1. Axenfeld: Dystrophien des Korneaparenchyms, Klin. Monatsbl. f. Augenh. **85**:493 (Oct. 1930; Lehrbuch der Augenheilkunde, Jena, Gustav Fischer, 1921.

2. Fuchs, E.: Localization of Pathologic Changes in the Cornea, Arch. Ophth. **3**:588 (May) 1930.

3. Groenouw: Arch. f. Augenh. **21**:281, 1890.

4. Gifford: Am. J. Ophth. **9**:81 (Feb.) 1926.

5. Spanlang: Clinic and Pathology of Dystrophia Adiposa and Congenital Dyskeratosis, Ztschr. f. Augenh. **62**:21 (May) 1927.

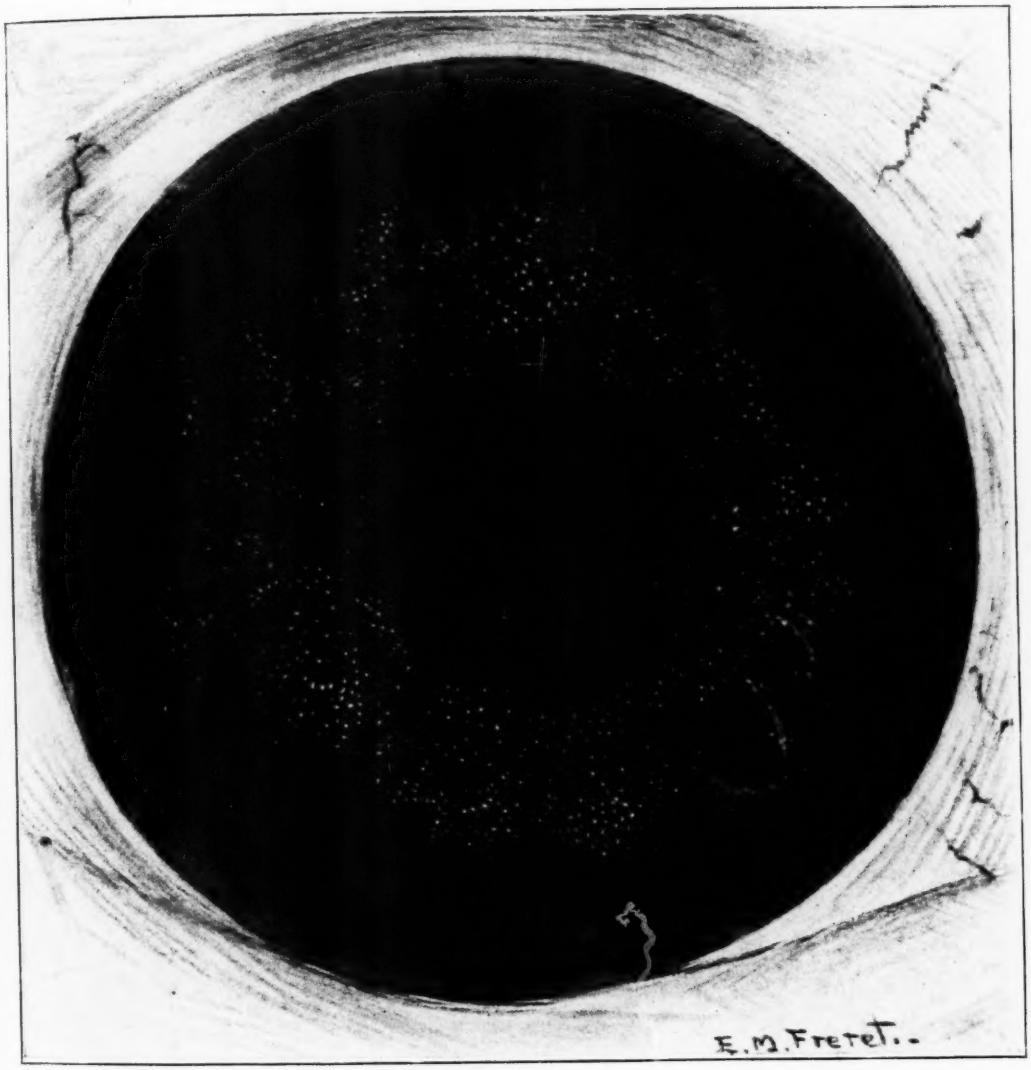

Fig. 3.—A colored drawing of the corneal opacity as seen under the slit-lamp.

cornea was removed and stained; examination proved that the spherical masses were cholesterol fat globules. The blood of the patient had a high cholesterol content. Spanlang mentioned that juvenile arcus senilis was produced experimentally by Joel by increasing the cholesterol in the blood of rabbits. Meyer [6] described a case of dystrophia adiposa in a woman, 65 years of age. The cornea was the only place in the body in which the needle-like crystals of cholesterol could be observed intra vitam. With high magnification they appeared as oblong, shiny, refractile bodies. The examination of the trephined cornea with polarized light showed cholesterol and cholesterol esters. The blood serum of the patient had a high lipoid content. Kraupa [7] reported a case of "ichthyosis corneae" in which fine, round, grayish dots resembling sand could be seen with the corneal microscope in the epithelium and Bowman's membrane. These trophic disturbances were also present in the skin. In keratitis punctata superficialis associated with nervous leprosy (Pillat [8]), the grayish dots are located in the superficial layers of the cornea. No evidence of any systemic disease was present in my patient.

In reviewing the literature on congenital corneal opacities, the Krukenberg type of pigmentation is worthy of consideration, because a few dots in this case were of a brownish hue. In 1899, Krukenberg [9] reported three cases of congenital pigmentation of the deep layers of the cornea with a peculiar arrangement of a spindle-shaped granular pigment. He named it "congenital melanosis corneae," and attributed it to the approximation of the pigment of the iris or pupillary membrane to the cornea at a certain stage of embryonic life, before the formation of the anterior chamber (sixth week). It could occur in brown eyes only, because the pigment was of the same color as that of the iris stroma. On the other hand, Stock [10] described a case showing gray dots instead of brown (the patient had gray irides). In his opinion the cells remaining on the posterior surface of the cornea after the formation of the anterior chamber possess the power of developing pigment at the time of birth. Korobova [11] found spindle pigmentation in an

6. Meyer: Beitrag zum Krankheitsbild der dystrophischen Adiposa korneae, Klin. Monatsbl. f. Augenh. **81**:786, 1928.

7. Kraupa: Die Ichthyosis der Hornhaut, Klin. Monatsbl. f. Augenh. **65**: 903, 1920.

8. Pillat, A.: Leprosy Bacilli in the Scraping from the Diseased Cornea in a Leper, and Comments on Keratitis Punctata Superficialis, Arch. Ophth. **3**:306 (March) 1930.

9. Krukenberg: Beiderseitige angeborene Melanose der Hornhaut, Klin. Monatsbl. f. Augenh. **37**:254, 1899.

10. Stock: Klin. Monatsbl. f. Augenh. **34**:770, 1901.

11. Korobova: Russk. Oft. J., April, 1929, vol. 9.

eye that she removed because of uveitis. Microscopic examination of the slides showed degenerative and proliferative changes; she expressed the belief, therefore, that the Krukenberg spindle is of inflammatory origin. Edgerton [12] gave a summary of the literature on the congenital opacity of the Krukenberg type. Thirty-six cases have been reported since 1899. In all instances the spindle was vertical or oval, and located in the deep layers of the cornea. Nearly all cases were bilateral and associated with myopia, with the exception of four in which there was hyperopia. Most of the patients had brown irides. The only feature that my case had in common with the Krukenberg type was the brown color of the iris and a few dots with a brownish hue, but none of the other characteristics of so-called melanosis corneae were present. In 1910, Mohr [13] found only eight cases of congenital corneal opacities among 60,000 patients (since no slit-lamp existed then, many cases may have been overlooked).

Seefelder [14] stated that there are two types of congenital opacities of the cornea, one due to disturbance in development and the other produced by inflammatory changes. Usually there are bilateral central opacities of the cornea resembling keratitis parenchymatosa, and associated with defects in the deep layers of the cornea. Peters [15] considered all congenital opacities of the cornea to be an anomaly of development, caused by penetration of the aqueous through defects in Descemet's membrane. A case of unilateral pigmentation of the right cornea was reported, in which defects of Descemet's membranes were observed under the microscope (Maschimo [16]). There was no evidence of malformation in my case.

The only picture that I have been able to find similar to the one that I am reporting, was described in 1925 by Key [17] under the title, "Congenital Zonular Opacities of the Cornea." In his case, however, there was a crayon-like line surrounding the opacities, and also a high lipoid content of the blood, whereas my patient had a normal blood cholesterol content.

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12. Edgerton, A. E.: Krukenberg's Spindle, Arch. Ophth. **3**:599 (May) 1930.

13. Mohr: Beiträge zur Frage des Entstehung der kongenitaler Hornhaut Trübungen, Klin. Monatsbl. f. Augenh. **10**:338, 1910.

14. Seefelder: Die Missbildungen des menschlichen Auges, Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930.

15. Peters: Die angeborenen Fehler und Erkrankungen des Auges, Klin. Monatsbl. f. Augenh., 1906.

16. Maschimo: Ein Beitrag zur Kenntnis der angeborenen Hornhaut-Trübungen, Klin. Monatsbl. f. Augenh. **71**:184 (Sept.) 1923.

17. Key, Ben Witt: Congenital Zonular Opacities of the Cornea, Am. J. Ophth. **8**:97 (Feb.) 1925.

CONCLUSION

A careful review of the literature shows that this corneal lesion does not resemble the various types of corneal dystrophies. It could not be linked to any of the systemic diseases, as the laboratory tests and the general physical examination gave negative results. The restriction of this opacity to the cornea of one eye only, the absence of other congenital abnormalities and the history of an injury make trauma a possible causative factor, but the clinical appearance of the cornea does not bear out this suggestion. A congenital origin of this lesion is much more probable, because of the symmetrical and regular distribution of the granules in the cornea. The limitation of the opacity to the parenchyma may indicate that it occurred during the cleavage of the mesodermic tissue. The microscope may some day reveal the true nature of this lesion, which at present is still open to conjecture.

MEDULLATED NERVE FIBERS ASSOCIATED WITH CHOROIDITIS

REPORT OF A CASE WITH PRELIMINARY STUDIES ON THE CAUSE OF THE APPEARANCE OF MEDULLATED NERVE FIBERS IN THE RETINA *

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Cases of extensive medullated nerve fibers in the retina are rare. The case reported here is particularly interesting because of the association with choroiditis. So far a similar case has not been reported in the literature.

In addition I have studied specially stained preparations of optic nerve and retina in an endeavor to account for the medullated nerve fibers. The results of this investigation led to the conclusion that the anomaly of these fibers in the retina may be due to the abnormal presence of the myelin-forming elements (the oligodendroglia) in the layer of nerve fibers.

REPORT OF CASE

A man, aged 35, came to the Cornell Clinic on Nov. 15, 1930, complaining of loss of sight in the right eye. He stated that it was his custom to have a yearly examination, and that with the exception of an occasional change in glasses, he had never had any trouble with his eyes until September, 1930, when he noticed progressive blurring of vision in the right eye. The family and past personal histories were unimportant. The vision in the right eye was 10/200 unimproved; in the left eye, 20/200 improved to 20/30. Under a cycloplegic, the error of refraction was -3 D. $=$ 1 D. $\times$ 180 degrees in each eye. Examination of the anterior part of the right eye with the slit-lamp did not reveal a pathologic process, except that in the vitreous there were considerable contortion and disarrangement of the layers, which showed increase in density. In the vitreous there was a marked abundance of granules of pigment, the reddish brown of which suggested origin in the retina rather than in the blood. With the ophthalmoscope the vitreous showed numerous delicate floating opacities, and in the fundus there were extensive areas of opaque nerve fibers, particularly on the nasal side of the nerve. These fibers extended above and below on this side, far into the periphery. The less numerous opaque fibers temporally made a wide arc, leaving the macula and the adjoining retina free. The region of the macula was hazy and edematous, obscuring the normal reflexes to light. The blood vessels appeared to be normal. In places they were lost, lying deep under opaque fibers. In still other areas, where they were situated more superficially, they could be seen indistinctly throughout their course. In the far periphery, distinct striations of the opaque

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* Read before the New York Academy of Medicine, Section of Ophthalmology, Jan. 19, 1931.

fibers could be made out. Centrally, the nerve fibers appeared as an almost homogeneous grayish layer, darkly stippled, resembling the areas of fatty degeneration sometimes seen in albuminuric retinitis. In general, the direction of the opaque fibers corresponded with that of the blood vessels. In the far periphery of the lower nasal quadrant, there was a small area of retinal detachment. No tear was visible.

Examination of the anterior part of the left eye with the slit-lamp gave negative results, and the structure of the vitreous showed little disorder of its layers. There was a slight amount of pigmentation similar to that in the right eye. With the ophthalmoscope, there were a few delicate opacities in the vitreous. Opaque nerve fibers were seen in the fundus of this eye, but in a lesser degree than in the right eye, and there were no foveal changes. The opaque fibers extended from the nerve head nasally above and below to the far periphery and, in the main, followed the general direction of the vessels. In the temporal region they arched widely, leaving the macula and the adjoining retina free. Above, the vessels were visible throughout their course. In the lower field the blood vessels were almost completely hidden, their terminal branches being visible distally. It was rather difficult to take the field of either eye accurately because of the fleeting changes in the vision. This was no doubt due to the fact that there was some retinal sensitivity in the areas of the medullation, even where this was densest. In the right eye, with a 5 mm. white test object, there was a complete loss of the temporal field. Nasally, there was a contraction of about 10 degrees. In addition, there was an absolute central scotoma. In the left eye there was a uniform concentric contraction of from 5 to 10 degrees. The blind spot in the left eye was slightly enlarged. The intra-ocular tension was normal in both eyes.

The diagnosis was: in the right eye, medullated nerve fibers associated with a low grade central choroiditis and with a small peripheral retinal detachment; in the left eye, medullated nerve fibers with a few opacities in the vitreous. Complete physical examination revealed only a right upper molar tooth in which a pathologic process was suspected. The Wassermann and tuberculin tests proved negative, as did a roentgenogram of the head. The tooth was extracted, and an apical abscess was found. Four days later, the patient noticed a decided improvement in the vision in the right eye, which increased to 20/70. The absolute central scotoma became relative. The edema in the region of the macula subsided, but there still remains a slight disturbance of the layer of pigment. Since then there has been no further improvement in the vision or in the clearing of the opacities in the vitreous. Throughout this time there has been no change in the condition of the left eye.

COMMENT

Opaque nerve fibers were demonstrated first by Virchow [1] in pre-ophthalmoscopic days. Von Hippel [2] stated that the medullation occurs from the center toward the periphery; that the optic nerve is the last of the cranial nerves in which it is completed, and that it reaches the lamina cribrosa after birth. New-born rabbits likewise present no trace of medullated fibers extending directly from the papilla. The

1. Virchow: Zur pathologische Anatomie der Netzhaut, Virchows Arch. f. path. Anat. **10**:170, 1856.

2. von Hippel, E.: Sind die markhaltigen Nervenfasern eine angeborene Anomalie? Arch. f. Ophth. **49**:591, 1900.

medullary sheaths in this animal are not fully developed until the tenth day after birth. Their appearance after birth caused von Hippel to say that opaque fibers are not congenital. Later, Sattler [3] examined a series of human fetuses from 5½ to 10½ lunar months old. He stated that medullations begin in the central nervous system and spread peripherally along the nerve. According to him, there are no medullations at 5 months. At 7 months, medullary sheaths may be traced from the optic tract to the beginning of the optic nerve, and by term these reach the lamina cribrosa. He further stated that the medullation appears first as minute droplets of a lecithin-containing substance in the protoplasm of the glial cells, and that these droplets run together and surround the axis cylinders. The sheaths are very thin, and as development proceeds, they thicken, proximally at first, so that at birth the medullary sheaths in the optic tract are thicker than those in the nerve behind the eye, whereas in the adult, they are of the same thickness.

Most authorities state that the anomaly is due to the fact that the medullary development exceeds its customary limits by passing through the lamina cribosa and investing the nerve fiber layer of the retina. According to the histologic observations of others and to my investigations, this does not appear to be true, because in no case have the medullated fibers been found passing through the lamina cribrosa to any extent. The medullary sheaths stop rather abruptly at the proximal border of the lamina cribrosa and may recur in the retina. It seems as if the lamina blocks the progress of the medullary sheaths. This agrees with the fact that in animals' eyes, which are normally without a lamina cribrosa, the medullary sheaths pass uninterruptedly from the optic nerve to the retina. Piersol [4] believed that the medullation of a nerve fiber is due to a sheath of delicate reticular framework composed of neurokeratin. As a matter of fact, medullated nerve fibers in the optic nerve proper, and in their anomalous location in the retina, have no neurilemma or sheath of Schwann. Gradle [5] stated:

> The cause of sporadic medullation of retinal nerve fibers falls among still unexplored phases of ophthalmology. . . .

He concluded:

> First, the enlargement of the blind spot found in such cases seldom corresponds in size or shape to the ophthalmoscopic picture of the medullated area; second, the disparity between the size and shape of the blind spot and the ophthalmoscopic picture of the mass of medullated nerve fibres within the retina, are

3. Sattler: Ueber die Markscheidenentwicklung im Tractus opticus, Arch. f. Ophth. **90**:296, 1915.

4. Piersol, G. H.: Human Anatomy, Philadelphia, J. B. Lippincott Company, 1907.

5. Gradle, Harry S.: Blind Spot: III. The Relation of the Blind Spot to Medullated Fibers in the Retina, J. A. M. A. **77**:1483 (Nov. 5) 1921.

Fig. 1.—Fundus of the right eye, showing extensive areas of medullated nerve fibers, small retinal detachment in the periphery and changes in the macular region.

Fig. 2.—Fundus of the left eye, showing extensive areas of medullated nerve fibers.

not opaque to the incident light for the entire length of the medullation, and the degree of opacity cannot be deduced from the ophthalmoscopic picture; third, in such cases the enlargement of the blind spot indicates merely the amount of retinal area that is prevented from functioning by the presence of overlying medullated nerve fibres in a layer sufficiently thick to obtrude incidental light.

The foregoing statements are entirely in agreement with the findings in my case. In the right eye, the entire temporal field was lost. This was due not only to the presence of the medullations, but to the choroiditis, and also to the retinal separation in the far periphery. It was interesting also to note that over the small area of separation in the far periphery the medullations were not affected. In the left eye, despite the extensive area affected, there was only a slight enlargement of the blind spot and a small degree of concentric contraction of the fields. Lloyd [6] stated that the blind spots are definitely enlarged in all cases in which the medullations extend from the disk, and advised using the stereoscopic method or a two point fixing plan for the monocular method. He also suggested making the test more sensitive by using a test object subtending a very small visual angle, by reducing the brightness of the test object, or by subduing the illumination by the introduction of colored glass, preferably red. Opaque fibers are often seen in eyes associated with myopia and with choroidal colobomas. Bachman [7] described a case with embolism of the central retinal artery. Jackson [8] reported a case of medullated nerve fibers with uveitis. Three cases of opaque fibers of the macular region were described by Soriano,[9] Libby [10] and Pascheff,[11] respectively. In atrophy of the optic nerve, the opaque fibers disappear. Wagenmann [12] was the first to describe such a case. Later, Metzger [13] reported another. In twelve cases of Recklinghausen's disease, Fischer [14] found four in which there were medullated nerve fibers.

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6. Lloyd: Visual Field Studies, personal communication.

7. Bachman: Schwund Markhaltigenervenfasern in der Retina nach Embolie der Arteria centralis retinae, Arch. f. Ophth. **107**:10, 1921.

8. Jackson, E.: Uveitis with Opaque Optic Nerve Fibres, Am. J. Ophth. **1**:448, 1918.

9. Soriano: Medullated Fibres of the Macular Area, Klin. Monatsbl. f. Augenh. **70**:220, 1922.

10. Libby, G. F.: Medullated Nerve Fibres Involving the Macula, Am. J. Ophth. **8**:713, 1925.

11. Pascheff: Medullated Nerve Fibres Involving the Macula, Am. J. Ophth. **9**:65, 1926.

12. Wagenmann: Schwund markhaltiger Fasern bei Tabes, Arch. f. Ophth. **11**:456, 1894.

13. Metzger: Atrophie markhaltiger Nervenfasern, Klin. Monatsbl. f. Augenh. **72**:801, 1924.

14. Fischer, H.: Beiträge zur Recklinghausenschen Krankheit, Missbildungen am Auge, besonders die markhaltigen Nervenfasern der Netzhaut, Dermat. Ztschr. **42**:143, 1924.

The case reported here is interesting, first because of the wide extent of the medullated nerve fibers; second, because of the association with choroiditis, retinal detachment and opacities in the vitreous, and third, because it confirms Gradle's observations that the scotomas and the enlargement of the blind spots resulting from the presence of opaque nerve fibers are always smaller than one would expect from the picture seen in the fundus.

AN EXPLANATION FOR THE PRESENCE OF MEDULLATED NERVE FIBERS IN THE RETINA

Until recently no systematic study of the various types of glial cells in the optic nerve and retina had been made, nor had any attempt been made to explain their relationship to the presence of medullated nerve fibers in the retina. There are three types of glia cells in nerve tissue: astroglia, microglia and oligodendroglia. The astroglia, of two types, namely, protoplasmic and fibrous, are found throughout the nervous system. This type of neuroglia is characterized by the fact that the cells have some prolongations, the ends of which are applied against the walls of the small vessels. The microglia, in all probability mesodermic in origin, are mostly phagocytic, and correspond to the histiocytes of the connective tissue system. The oligodenroglia are seen in rows and are especially conspicuous in the white matter, where their prolongations envelop the nerve fibers. These cells have to do with the formation of the medullary sheaths of the nerve fibers. Hortega [15] stated that the secretion from the oligodendroglia forms the medullary sheaths. In Schilder's disease, the oligodendroglia are swollen and filled with a mucoid substance. As a result, there are a fragmentation and a loss of the myelin sheaths.

The three types of glia cells are present in the optic nerve and can be demonstrated by selective staining methods. Figure 3 shows two large astroglia cells in the human optic nerve stained by Hortega's silver carbonate method (formula for astroglia). Figures 4 and 5 show the microglia and oligodendroglia, respectively. In figure 4, the characteristic shape of the microglia cells is clearly seen. The microglia elements in the optic nerve, as is the case of the central nervous tissue, are evenly distributed, and in ordinary slides their nuclei can readily be identified on account of their irregular shape, small size and deeply stained karyoplasm (fig. 3, *m*).

The oligodendroglia nuclei form the well known neuroglia rows. With the method of Hortega each cell shows a dark cytoplasm from

15. del Rio Hortega, P.: Mem. r. Soc. españ. de hist. nat. **14**:1, 1928.

which a few prolongations emerge—hence the term "oligodendroglia" given to these elements (fig. 5). Their prolongations penetrate between the nerve fibers, where they probably end as in the central nervous system, namely, forming rings and spirals around the fibers.

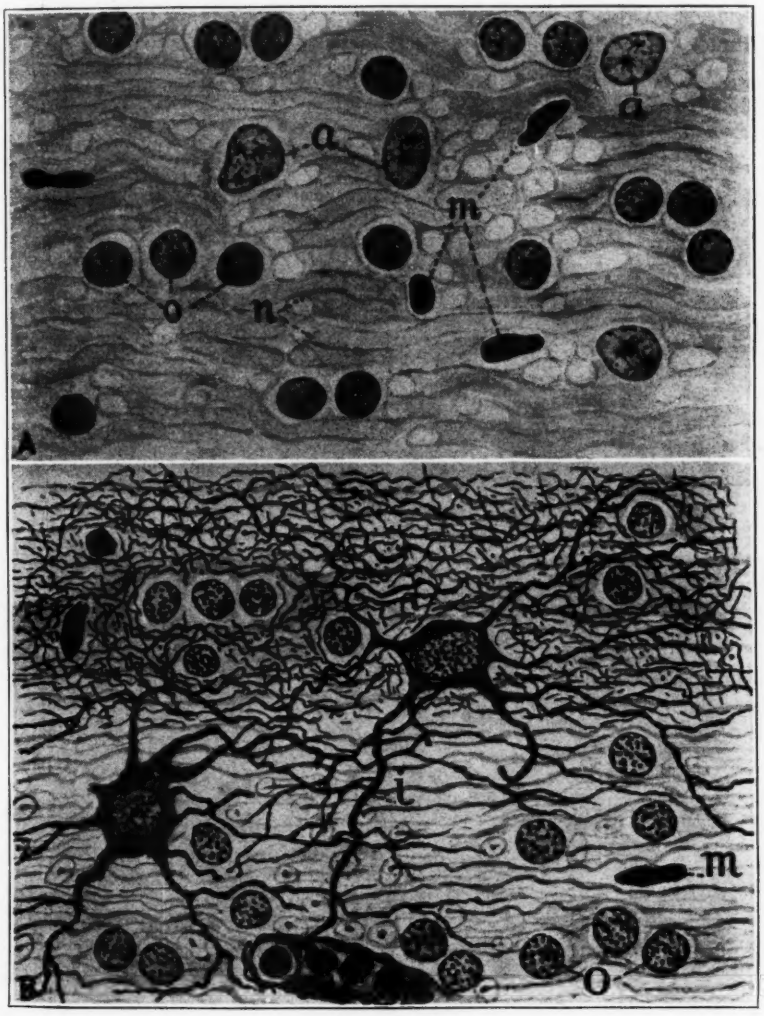

Fig. 3.—*A*, nuclei of the different types of neuroglia cells in the human optic nerve. Hematoxylin-eosin. *B*, two large astroglia cells in the human optic nerve. Hortega stain (for astroglia). The fine plexus of glia fibers has been omitted in the lower half of the drawing. *a* indicates astroglia nuclei; *i*, prolongation ending on the wall of a capillary; *m*, microglia nuclei; *n*, nerve fibers, and *o*, oligodendroglia nuclei. (Preparation and drawing by J. F. Nonidez.)

The presence of oligodendroglia in the retina has not been demonstrated. In all the preparations of normal retina that I studied, there

Fig. 4.—Microglia in a longitudinal section of the optic nerve. Hortega stain (for microglia). *A, B, C* and *D* indicate microglia cells; *E*, oligodendroglia; *F*, nerve fibers, and *G*, connective septums. (After Lopez Enriquez.)

was no evidence of any of the characteristic rows of oligodendroglia. The existence of astroglia has been known since the studies of Cajal,[16] whose text contains a good illustration showing the astrocyte in the retina, lamina cribrosa and optic nerve, stained by the silver chromate

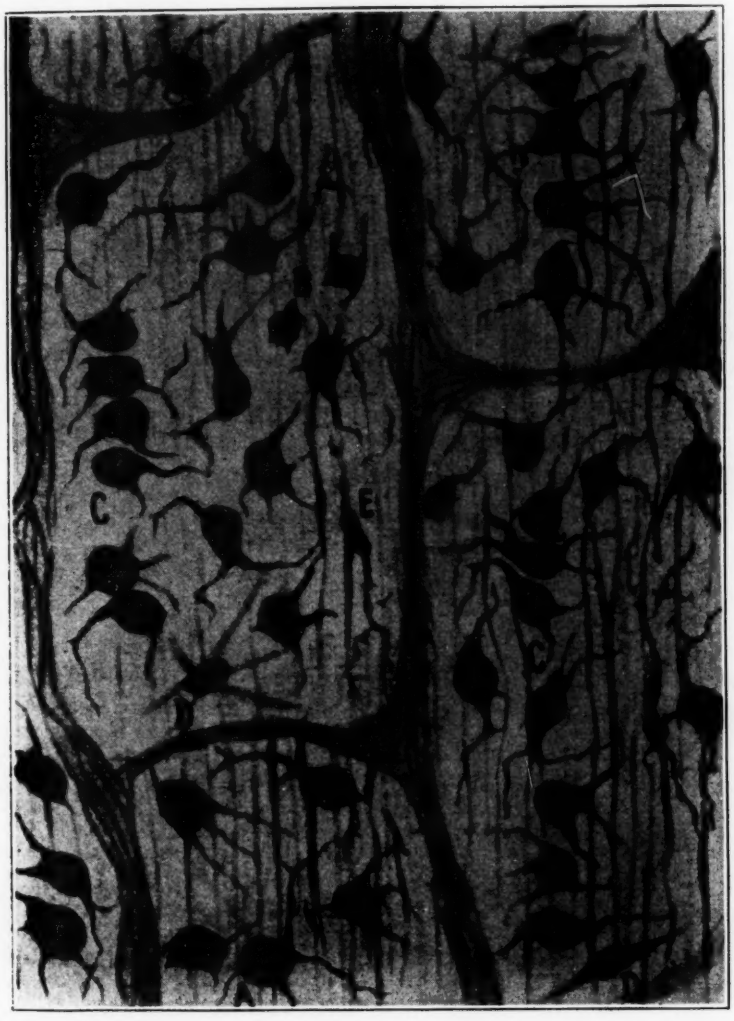

Fig. 5.—Oligodendroglia in a longitudinal section of the optic nerve. Hortega stain (for oligodendroglia). *A, B* and *C* indicate oligodendroglia cells; *D*, fibrous astroglia; *E*, microglia, and *F*, connective septums. (After Lopez Enriquez.)

method of Golgi. On the other hand, the presence of the microglia has been shown by Lopez Enriquez.[17]

16. Ramón y Cajal, S.: Histologie du système nerveux, Paris, A. Maloine, 1911, vol. II, p. 302, fig. 189.

17. Lopez Enriquez, M.: Bol. r. Soc. españ. de hist. nat. **26**:294, 1926.

Without denying altogether the possible presence of oligodendroglia, it can safely be said that the rows of oligodendroglia nuclei, so characteristic of the optic nerve, do not exist in the layer of nerve fibers of the retina, not even toward the papilla where this layer is rather thick. The normal picture in slides stained with hematoxylin and eosin is similar to that copied in figure 6. Most of the nuclei present belong to the cells of Müller, the astroglia (*a*) and the microglia (*m*). Rarely could a few isolated nuclei be interpreted as resembling oligodendroglia (*o ?*).

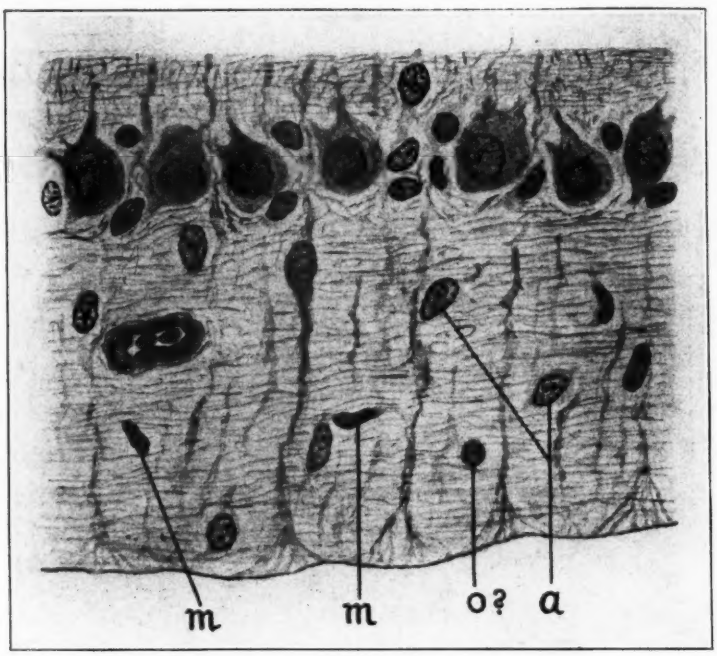

Fig. 6.—Normal human retina. Hematoxylin-eosin. Only the nerve fiber layer and the layer of ganglion cells have been represented. *a* indicates the astroglia nuclei; *m*, microglia nuclei; *o ?* resembles a nucleus of an oligodendroglia cell.

Around and between the ganglion cells the nuclei are more numerous, suggesting that they may belong to neuroglial satellites.

It would be interesting to know the distribution of the nuclei in retinas containing myelinated fibers. Unfortunately, material of this type is rare and is usually prepared with Weigert's myelin stain. This, while showing clearly the presence of myelin, does not disclose the distribution or character of the nuclei. If it is true that the medullated sheaths are formed by a secretion from oligodendroglia cells, it would be easily understood why the nerve fibers in the retina, which have no oligodendroglia, are nonmedullated; and likewise, why the anomalous

presence of oligodendroglia in the layer of nerve fibers in the retina would result in medullated retinal nerve fibers. In another communication I hope to show that the myelinization of the nerve fibers in the retina is definitely correlated with the presence of abundant oligodendroglia.

SUMMARY

1. Three types of glia cells can be demonstrated in the optic nerve: astroglia, microglia and oligodendroglia.

2. Only two types, microglia and astroglia, have been found in the nerve fiber layer of the retina.

3. It appears that the rows of oligodendroglia investing the nerve fibers in the optic nerve cease abruptly at the proximal margin of the lamina cribrosa.

4. According to the investigations of Hortega, the secretion from the oligodendroglia seems to form the medullary sheaths.

5. In the retina, the abnormal presence of oligodendroglia in the layer of nerve fibers may be responsible for the medullated nerve fibers.

METASTASIS IN THE SHEATH OF THE OPTIC NERVE FROM CARCINOMA OF THE STOMACH *

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AND
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That metastatic deposits of carcinoma in the eye affect the choroid in most instances is a fact observed clinically and confirmed pathologically. Behr [1] stated that in 65 per cent of cases the primary source is the breast, and he cited only four instances up to 1922 in which the nerve alone was involved. When both were affected, it appeared that the metastasis to the choroid had broken through into the nerve. In his own case, however, separate foci were found, so as to suggest distinct emboli of carcinoma to choroid and nerve.

Few cases of metastatic involvement of the optic nerve are reported in the literature. Abelsdorff [2] indicated the sheath of the nerve as being the initial resting place of such foci, from which the nerve itself was first compressed and then invaded by the new growth. This would appear to be the case especially in those instances in which the meninges and the intracranial portion of the nerve were implicated in the secondary growth. Isolated foci in the nerves have been noted, however, as in Elschnig's [3] case of bronchial carcinoma.

In addition to those of carcinoma of the breast, foci of bronchial adenocarcinoma (Ginsberg [4]) and carcinoma of the kidney (Elschnig [3]) have been observed on the sheath of the optic nerve. The ability of tumor cells in the lung to break into blood vessels is well known. In the cases reported in which a gastric carcinoma was the primary tumor

* Submitted for publication, March 21, 1931.

* From the Laboratories and Ophthalmological Division, Mount Sinai Hospital.

1. Behr: Metastatische Karzinose der Choroidea und des Sehnerven, Klin. Monatsbl. f. Augenh. **79**:788, 1922.

2. Abelsdorff: Metastatischen Karzinom des Sehnerven, in Henke and Lubarsch: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 11, p. 808.

3. Elschnig: Die metastatische Geschwülste des Sehorgans, Arch. f. Augenh. **22**:149, 1891.

4. Ginsberg: Doppelseitige Sehnerven Metastasen eines bronchial Karzinom als Ursache völliger Erblindung, Klin. Monatsbl. f. Augenh. **67**:232, 1921.

(Saxer,[5] Marchand,[6] Cords[7]), the meninges at the base of the brain were involved as well. Marchand reported infiltration into the sacral nerve roots, suggesting a direct course to the meninges through the subarachnoid space by way of the spinal canal.

Tumor in the sheath or nerve in all cases led to some loss of vision and even to complete amaurosis. Pallor of the nerve and papilledema were variable signs. Amaurosis was more complete when the intracranial portion of the nerve was embedded in meningeal metastasis. Under such

Fig. 1.—Metastatic carcinoma in the sheath of the optic nerve.

conditions papilledema could be attributed as much to a general increase of intracranial pressure as to obstruction by tumor in the sheath of the optic nerve.

The case to be reported is of interest because of the total absence of visual disturbance and ophthalmoscopic signs of a growth in the sheath of the nerve.

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5. Saxer: Verhandl. d. deutsch. path. Gesellsch. in Karlsbad, 1902.

6. Marchand: Ueber diffüse Verbreitung von Karzinomzellen in den Meningen von einem Magenkarzinom aus, München. med. Wchnschr., 1907, no. 13, p. 636.

7. Cords: Karzinose der Optikus, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **43**:293, 1922.

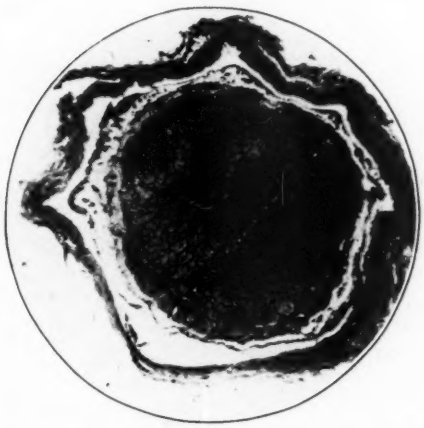

Fig. 2.—Section of nerve 10 mm. behind the lamina cribrosa. *A* indicates the infiltrated arachnoid.

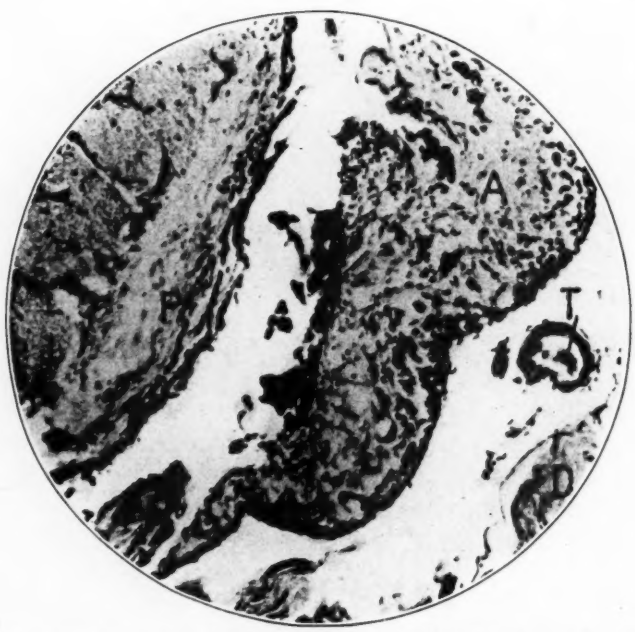

Fig. 3.—Section of nerve about 10 mm. behind the lamina cribrosa. *P* is the pia infiltrated by cords of carcinoma cells; *A*, markedly thickened arachnoidal sheath invaded by carcinoma; *D*, the dura, and *T*, a small artery containing tumor cells.

REPORT OF A CASE

M. S., a man, aged 48, was admitted to the medical wards with a diagnosis of carcinoma of the stomach. Numerous evidences of metastasis were noted. The fundi were normal. As far as could be ascertained, there were no visual disturbances. Death occurred one month following admission.

At autopsy the observations were: ulcerating carcinoma of the stomach, metastases to the peripancreatic, peritracheal, periaortic and cervical lymph nodes, and to the skin, liver, kidneys, intestines and gallbladder.

Fig. 4.—Enlargement of artery with tumor cells, shown in figure 3.

Fig. 5.—Alveolar arrangement of carcinoma cells (*AL*) in the arachnoid (*AR*).

Microscopic examination showed the primary tumor to be adenocarcinoma of the stomach. Many tumor cells were seen in the blood vessels of the lungs, spleen and pancreas and in the glomeruli of the kidneys.

The right eye, with 11 mm. of nerve attached, was fixed in Bouin's fluid and section in paraffin. Three millimeters of the nerve was cut separately in cross-section. The brain was not examined; the state of the meninges was therefore not determined.

There was no evidence of carcinoma in any portion of the uvea or in the disk. The latter showed no swelling.

In longitudinal section (fig. 1), the new growth stood out as a dark-staining mass of tissue between the nerve and the dura, and extending to the apex of the vaginal space. Its thickest portion was from 4 to 5 mm. behind the cribriform

plate. In cross-section (fig. 2), the coats of the nerve were more distinct, and the tumor not so dense. Here it was clear that the dura was entirely free from infiltration, and that the arachnoid bore the burden of the new growth. Its trabeculae were markedly swollen. At some points (fig. 3), this membrane was thickened to five or six times its normal size. The tumor cells occurred singly and in clumps and surrounded the blood vessels intimately. Some were as large as giant cells. Most striking was the presence of a clump of tumor cells within the lumen of an arteriole in the subdural space (figs. 3, 4 and 5). In places, the cells were so disposed as to duplicate the tubular arrangement of the alveolar carcinoma in the stomach (fig. 5).

The pia in its entire circumference contained cords of cells similar to those in the arachnoid. This infiltration was more uniform than in the overlying arach-

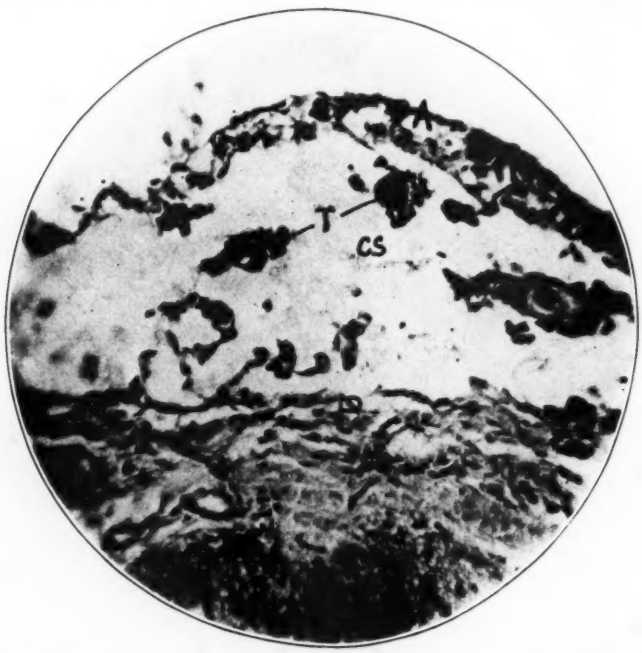

Fig. 6.—Masses of carcinoma cells (*T*) free in the subarachnoid space; *A*, arachnoid; *CS*, precipitated elements of the cerebrospinal fluid; *P*, pia.

noid. Figure 6 denotes a probable mode of pial involvement. Throughout the subarachnoid space were clumps of tumor cells detached from the arachnoid and lying free among faintly staining precipitated elements of the cerebrospinal fluid. Some of these were in contact with the pia.

There was no evidence of tumor in the substance of the nerve. Staining with Weigert's stain for medullary sheaths failed to show evidence of atrophy in any portion of the nerve available for study.

COMMENT

The presence of tumor cells in the blood vessels of the lungs and in the arteries of the spleen and kidneys proves the distribution of metastases by way of the blood stream. A similiar situation in the optic

nerve sheath suggests strongly that the tumor was brought to the arachnoid by the blood stream. This membrane bears the brunt of the metastasis. It is not inconceivable that the pia was at least partly infiltrated through detached clumps in the subarachnoid space.

Growth in the sheath of the nerve is compatible with the absence of papilledema or other ophthalmoscopic signs, especially if the growth is gradual and the intracranial portion of the nerve is free. The lack of visual disturbance is distinctive in this case. It is most easily explicable, of course, by the possibility that the sheath was invaded late in the disease. In this regard it is interesting to note the disparity between the state of vision and the condition of the nerve on section in cases previously reported. The amaurosis in Marchand's [6] patient was much greater than that indicated by the changes in the nerve. Similarly, in Holden's [8] case of carcinoma of the breast, there was a disproportion between the number of degenerated optic nerve fibers and the advanced degree of amaurosis.

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8. Holden: A Case of Metastatic Carcinoma of One Optic Nerve with Peculiar Degenerations of Both Nerves, Arch. Ophth. **31**:427, 1902.

POSTTRAUMATIC TUBERCULOUS UVEITIS*

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An effort will be made in this paper to discuss the relationship of trauma to tuberculous uveitis, and a review of an illustrative case confirmed by histopathologic studies will be given.

The relation of injury to the development of ocular tuberculosis has long been recognized. This applies not only to the eye, but to the chest, bones and joints. These injuries and their consequences are not without interest from the point of view of the responsibility of the employer for accident under the compensation law. Frequently the accident is said to be so slight as to escape the attention of the patient at the time. In nearly all cases, the tuberculosis following ocular injury is serious. Lloyd [1] and others have reported generalized miliary tuberculosis and death following tuberculosis of the eye after injury. It is of interest to note that in the majority of cases reported it occurs in persons who appear to be in good general health and in whom no other evidence of organic disease is readily recognizable.

In this type of case, the question is always asked as to whether the infection occurred at the time of injury, or whether, because of the lessened resistance, the area was secondarily infected from some distant focus. The distinction between the two types of infection, as pointed out by Stephenson,[2] is not without practical importance. For example, an exogenous lesion such as a tuberculous lacrimal sac may be removed completely, and all chance of generalization be thereby prevented. On the other hand, it would be of little value to remove an eye with tubercle of the iris or choroid with a view to preventing generalization, since the condition practically always originates from a tuberculous lesion elsewhere. It is quite another thing when removal of such an eye is rendered necessary by the local conditions, especially by pain.

Considerable experimental work has been done on the relation of tuberculosis to trauma. In 1878, Max Schueller [3] injected human tuber-

* Submitted for publication, March 28, 1931.

* From the Department of Ophthalmic Surgery, University of Michigan.

1. Lloyd, R. I.: Tuberculosis of the Eye, Am. J. Ophth. **13**:753 (Sept.) 1930.

2. Stephenson, Sydney: Tuberculosis of the Eye, Lancet **2**:1530, 1913.

3. Schueller, K. H. M.: Experimentelle und histologische Untersuchungen über die Entstehung und Ursachen der scrophulösen und tuberkulösen Gelenkleiden; nebst Studien über die tuberkulöse Infection und therapeutischen Versuchen, Stuttgart, Ferdinand Enke, 1880, vol. 7, p. 8.

culous expectoration into the lungs of dogs and rabbits and then caused the knee joints of these animals to be sprained or otherwise injured, with the result that in nearly all of them a granular tuberculous inflammatory reaction developed; in healthy control animals, the same injuries did not produce lesions. Schueller concluded that tubercle bacilli and other micro-organisms found their way to the injured joints of the infected animals.

Finnoff's [4] experiments with animals tend to prove that infection does not occur through the unabraded corneal or conjunctival epithelium, which would lead one to think that infection in man was usually hematogenous or lymphogenous in origin. He expressed the belief that the focus is often in the peribronchial lymph nodes and is in accord with the general consensus, which is that the bacilli are carried by lymphocytes or by the lymph and eventually reach the venous system through the thoracic duct or by shorter lymphatic trunks directly into the veins. Those that are not arrested in the lungs are distributed centrifugally by the arterial system. If they remain caught in the finer capillaries along their course, they become possible sources of infection, provided they are living bacteria. The blood acts as a passive conveyor, and the whole tendency is for the bacilli to get out of the blood circulation. Their emergence is produced through the intermediary of wandering cells which carry them through the intima of the vessels; once they have penetrated this intima, they are again in the perivascular lymphatic system.

The cases of posttraumatic tuberculous uveitis that show no evidence of perforation evidently belong to the endogenous type of infection. Infections occurring in this manner are either produced by a lymphogenous or hematogenous dissemination from a distance, or are due to a lighting up of an unrecognized quiet focus already present in the anterior uveal tract. Stock,[5] by his experiments, found that large tuberculous foci may exist in the ciliary body for a long time without producing any marked symptoms of cyclitis, and also that the tubercle bacilli may be found in the iris after apparent healing of the original lesions.

The factor of injury is not necessary to the localization of the tuberculosis, although injury accelerates the progress of the disease when the traumatized area is already tuberculous. It is recognized that every other infection encourages tuberculosis, which again brings in the the factor of lowered resistance whether local or general. Slight

4. Finnoff, W. C.: Ocular Tuberculosis, Experimental and Clinical, Arch. Ophth. **53**:130 (March) 1924.

5. Stock, W.: Tuberculosis as Etiology of Chronic Inflammations of the Eye and Its Adnexia, Especially of Chronic Uveitis, Am. J. Ophth. **24**:237, 1907.

injuries, like contusions, are more often factors in tuberculosis than more extensive injuries, the latter possessing more reaction with a greater blood supply. Krusius [6] and Finnoff [4] found that tissues of the eye that are exposed to infection from without are relatively resistant to tuberculosis; therefore, endogenous infection involving the uvea would be considered a frequent form of ocular tuberculosis when one considers the small capillary loops in which the bacilli may become lodged.

The following case is a typical example of posttraumatic tuberculous uveitis confirmed by histopathologic studies. Unsuccessful attempts to

Fig. 1.—Low power magnification of the anterior segment of the eye showing the conglomerate tubercle encroaching on the anterior chamber and beginning perforation at the limbus. The lens is partly absorbed. Hematoxylin and eosin.

demonstrate the tubercle bacilli in section were made both in the laboratories of Finnoff and in those of Parker.

REPORT OF CASE

A boy, aged 3 years, entered the department of ophthalmic surgery of the University of Michigan in August, 1929, with a history of being struck in the right eye one and one-half months previously. Subconjunctival hemorrhage and

6. Krusius, H.: Zur experimentellen Tuberkulöse des Auges, Deutsche med. Wchnschr. **37**:1545, 1600 and 2127, 1911.

ecchymosis into the lids and nose resulted from the trauma but not loss of vision. Three weeks after the injury, the right eye became acutely inflamed, and the patient complained of some pain and cloudiness of vision. He was apparently in good general health before and since the injury. An uncle suffering with pulmonary tuberculosis lived with the family for two years before going to a sanatorium. One brother of the patient was said to have died of tuberculous meningitis.

Examination showed moderate circumcorneal injection; the cornea was clear. Deposits were not present on the posterior surface of the cornea. The anterior

Fig. 2.—Medium power magnification through the iris and conglomerate tubercle showing the definite tubercle formation with giant cells, etc. The involvement of the posterior surface of the iris is well shown. *A* indicates the iris; *B*, the conglomerate tubercle, and *C*, the vitreous. Hematoxylin and eosin.

chamber was very shallow, especially peripherally where the filtration angle appeared to be obliterated. The iris bulged anteriorly; its surface was irregular and showed areas of infiltration. There were numerous dilated newly formed blood vessels over its surface. The iris was markedly thickened. The pupil was small and inactive and was filled with exudate. There was a complete, annular, posterior synechia. The left eye was normal. Intra-ocular tension was normal

on examination with the fingers. Vision was absent in the right eye and was 5/6 in the left eye. Pulmonary roentgenograms revealed increased peribronchial markings, especially in both upper lobes, but no definite parenchymal lesion. The Kahn test of the blood was negative. General medical examination failed to reveal any pathologic process. The patient was placed on atropine and salicylates, but the condition did not improve. He was moderately distressed due to the photophobia and increased lacrimation but there was little pain. The anterior chamber continued to decrease in depth, and the surface of the iris later presented small nodular grayish-red elevations scattered irregularly over its surface, so that a diagnosis of posttraumatic tuberculous uveitis was made. Because of the patient's

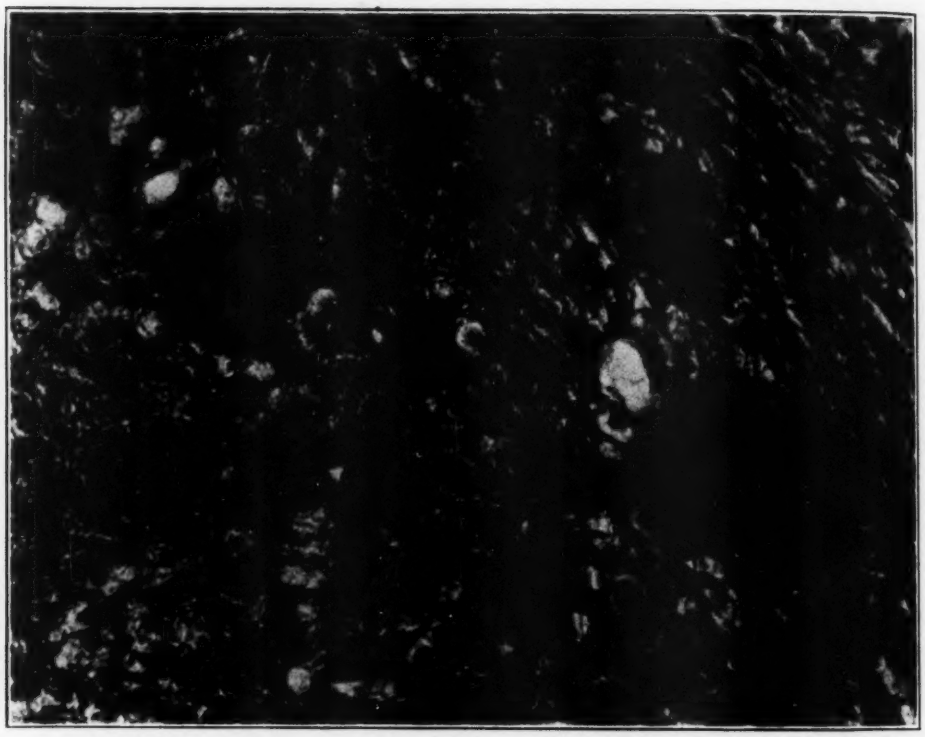

Fig. 3.—High power magnification of the tubercle showing multinuclear giant cells with peripherally placed nuclei, small round cell and plasma cell infiltration. Hematoxylin and eosin.

symptoms and the unfavorable prognosis, the eye was enucleated in December, 1929. Reexamination of the patient one year later failed to demonstrate the presence of tuberculosis in any form.

Grossly, the enucleated eye presented a conglomerate mass which nearly obliterated the anterior chamber (fig. 1). The mass was limited posteriorly by the hyaloid membrane and laterally by the ciliary bodies. The lens was partly absorbed and lay in a pocket in the posterior center of the mass. No gross lesions were seen in the vitreous, retina or choroid.

Microscopically, it is seen that the cornea presented a slight small-round cell infiltration throughout its entire extent (fig. 2 and 3) in the areas in which the granulation tissue was in direct contact with the cornea, the infiltration was more

marked. Descemet's membrane had been ruptured at some of these points, and the corneal stromal fibers here lost their parallel arrangement. Both limbi showed evidence of beginning perforation. Involving the entire base of the iris, the anterior extremities of the ciliary bodies and extending posteriorly to the hyaloid membrane, there was a large conglomerate mass of granulation tissue with a definite tubercle formation consisting of giant cells with plasma and small round cells surrounding them. The ciliary processes and the spaces between them were involved in this conglomerate mass. The ciliary bodies showed only small round cell infiltration. As is usually the case, the growth appeared to be developed largely from the posterior surface of the iris, pushing it forward as the conglomerate tubercle formed and extended (fig. 2). Numerous newly formed blood vessels were seen in the anterior surface of the iris as well as in its deeper stromal layers. It was markedly infiltrated with small round cells, plasma cells and phagocytes. The clump cells also appeared to be increased. The pigment epithelial layer had been broken up in part, some migrating with the newly formed granulation tissue posteriorly. The capsule of the lens was much wrinkled and showed evidence of perforation, as shown by the presence of granulation tissue between the capsule and the still unabsorbed lens. The cortical region of the remnant of the lens showed cataractous and vacuolar degenerative changes. All through the vitreous could be seen stringy opacities which radiated from the inflammatory tissue anteriorly. Clinging to these stringy vitreous opacities were numerous small round cells. The aqueous contained numerous small round cells and here and there small masses of fibrin. The retina and choroid were intact and were not involved.

CONCLUSION

Tuberculous uveitis may develop in an apparently healthy person as a result of trauma to the eye. As a result of the trauma, a latent tuberculosis is aroused into activity and a tuberculous lesion becomes localized at the site of the injury, the micro-organisms having been carried there from a focus elsewhere in the body, or injury has simply aggravated a lesion which was previously present in the uvea but giving no symptoms. The generally recognized omnipresence of tubercle bacilli in the human system and the part played by the condition of the body, as exemplified in general and local resistance, cannot be ignored, and it is here that injury by reducing resistance comes in as a possible factor in the development of the tuberculosis.

Clinical Notes

ENDOGENOUS PANOPHTHALMITIS ACCOMPANYING TONSILLITIS

Report of a Case *

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Endogenous panophthalmitis is fairly common, but careful search of the literature failed to bring to light a report of a case due to metastasis from the tonsils.

Probably the most common causative factor is puerperal sepsis, but cases have been reported accompanying cholecystitis,[1] prostatitis,[1] pneumonia,[2] measles,[3] mumps,[4] articular tuberculosis [5] and influenza.[6] The most recent literature on the subject is by Levine,[7] who reported a case due to B. coli from calculus pyonephrosis. Bacteriologically, the most common etiologic factors are the staphylococcus, streptococcus, pneumococcus and meningococcus.

REPORT OF CASE

Agatha O., aged 2, who was seen in consultation, had been suffering from tonsillitis for three days. The day before I saw her she had complained of pain in the right eye. The lids of that eye were edematous and tense; the cornea was hazy; the pupil was contracted, and the conjunctiva was inflamed, but there was no appreciable discharge. I advised hospitalization, and accordingly the next day she was admitted to the Manhattan Eye, Ear and Throat Hospital, the diagnosis being panophthalmitis.

The patient's general condition and that of her eye steadily became worse under routine treatment; the temperature ranged from 101.5 to 104 F., and there was some delirium. Examination of the nose failed to show any involvement of

* Submitted for publication, April 14, 1931.

1. Terrien, F.: Metastatic Ophthalmia, Ann. d'ocul. **159**:460, 1922.

2. Touzet: Pneumococcic Panophthalmitis, Arch. d'opht. **38**:372, 1921.

3. Fernandez, F. M.: Ocular Complications of Measles, Rev. cubana de oft. **3**:159, 1921.

4. Lundsgaard: Cyclitis with Parotitis, Klin. Monatsbl. f. Augenh. **57**:393, 1916.

5. Stock, W.: Metastatic Ophthalmia Through Tubercle Bacilli, Klin. Monatsbl. f. Augenh. **70**:356, 1923.

6. Samperi: Metastatic Panophthalmitis in Influenza, Gior. di. med. mil. **67**: 597, 1919.

7. Levine, J.: Metastatic Bacillus Coli Panophthalmitis from Calculus Pyonephrosis, Arch. Ophth. **3**:410 (April) 1930.

the sinuses. Roentgenograms of the sinuses showed moderate ethmoid involvement, especially on the left. Two days later, I performed an evisceration. The patient made an uneventful recovery.

Culture from the conjunctival sac showed a profuse growth of Staphylococcus albus and short-chain nonhemolytic Streptococcus mitis. Culture from the tonsils showed a profuse growth of Staphylococcus albus and short-chain hemolytic Streptococcus pyogenes. At the time of operation, culture from the interior of the globe showed the same bacteriology as the tonsils.

This case should, I think, emphasize the fact that the best and safest treatment for this condition is evisceration. Most cases of panophthalmitis in which the globe is left in situ result in phthisis bulbi. Early evisceration obviates the likelihood of involvement of the brain or the meninges by direct contamination through the foramen, the remaining sclera acting as a plug to prevent such transmission. Almost invariably when febrile symptoms are due to a condition of the eye, removal of that condition results in spectacular improvement. The ball of sclera with the muscles still attached serves as an excellent stump for a shell, and in the majority of cases good motility results.

The presence of the same etiologic factor in the throat and the interior of the globe and the absence of it, together with the presence of an entirely different organism in the conjunctival sac, led me to the conclusion that the condition of the eye was endogenous. The absence of any sign of involvement of the sinuses either by the x-rays or by direct nasal examination strengthens my conclusion.

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EPITARSUS *

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A case of epitarsus in an infant came to my attention through Dr. Kirschenbaum who saw the child at the Israel Zion Hospital soon after its birth. It was transferred to the Kings County Hospital, where I saw the patient through the courtesy of Dr. Moehle and Dr. Hallock. At the child's birth the corneas and scleras could be seen only by lifting the upper lids and a large roll of attached conjunctiva away from the eyeball. The normal rigidity of the upper lids imparted by the tarsal cartilage was missing. The mucous membrane of the upper lid usually in contact with the globe was swung forward with the hinge at the margin of the lid, and the mucous membrane extending from the upper tarsal margin into the usual fornix was redundant and prolapsed so that this tissue, usually tucked away high up in the recess of the upper fornix, was in contact with the eyeball. The left eye showed an even greater redundancy of the conjunctiva of the upper lid, and some of the tissue usually located in the fornix was rolled out and exposed along with the tarsal mucous membrane. In addition, at the inner angle of the con-

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junctival sac this redundant tissue continued on to the eyeball as a hypertrophied plica and from the eyeball on to the mucous lining of the lower lid, which was hypertrophied beyond the retrotarsal margin but not enough to show unless the lower lid was everted, when it rolled forward like the mucous membrane of the lower lid of an infant with ophthalmia neonatorum. The child did not open its eyes, but the corneas and scleras were normal. They could be seen only by raising the upper lid and then lifting the redundant tissue up and away from the eyeball. Birth had been normal, no forceps being used; labor had not been prolonged, and there had never been a discharge from either sac. Dr. Charles Hargitt removed the superfluous tissue, so that the eyes to outward appearances are now quite normal, but the child does not make any effort to open the eyes. Whether this function will appear later is a question, but at 1 month of age, the infant lies with eyes quietly closed as if asleep.

Fig. 1.—Epitarsus, soon after birth.

Epitarsus, congenital conjunctival apron, pterygium of the upper lid, tarsus duplex palpebrae and third eyelid are names used to describe an unusual congenital defect of the upper lid and its conjunctival lining. The lower lid is sometimes involved, and defects of the tarsal cartilage of the upper lid and even colobomas of the upper lid have been associated with it. A few cases of inversion of the upper lid are found in the records, which were due to cicatricial traction of the redundant fold (Denig, Wilde, Guerin, Berry, Stephenson, Harlan, Campbell). In most of the cases reported the condition was evident only when the upper or lower lids or both were everted, whereupon a fold of mucous membrane could be seen stretching from the upper fornix downward over the internal surface of the upper lid in the form of a triangle, the base being in the fornix and the apex or narrower margin attached to the mucous surface of the eyelid a short distance from the margin of the lid. A similar but less pronounced condition may also be found on the lower lid. The attachment at what was originally the free margin of the apron is believed to be due to accidental agencies, such as blenorrhea

or continued friction. The attachment may be much more extensive than this, but it is possible to insert a probe behind the fold to a greater or less extent.

The anomaly has been found in cases of trachoma and after croupous conjunctivitis, which has led some observers to call the condition pterygium of the upper lid, as they thought that the local irritation caused the development of an attached fold of conjunctival tissue which advanced from the fornix toward the margin of the lid, pulling a fleshy fold behind it. Thus Wibaut found twenty-four cases during the examination of many Amsterdam school children with trachoma; in twelve of these the condition was limited to the upper lid, in seven to the lower, and in five it affected both upper and lower lids. He has offered a reasonable explanation of the development of this strange condition which can be better appreciated after a glance at figure 4. From his point of view, the process of formation of the conjunctival sac and

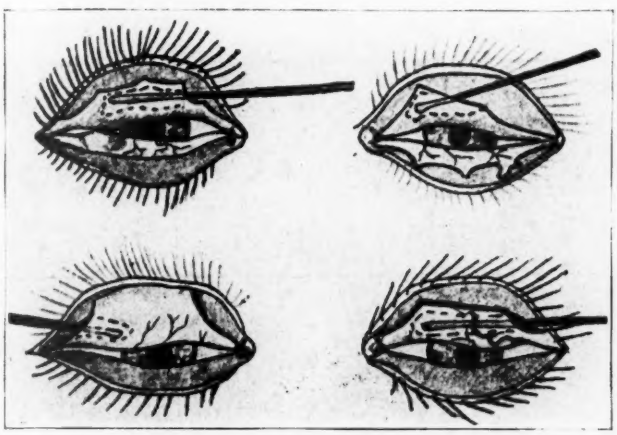

Fig. 2.—Epitarsus (Keutgen: Ztschr. f. Augenh. **45**:29, 1921).

the plica semilunaris in these cases is abnormal and the disappearance of the larger part of the original plical fold is arrested, leaving a redundancy of the upper and lower extensions of the plical fold. In the typical case, the fold hangs down from the fornix, like an extra lid with a free anterior and posterior surface. The present case departs from this type in that there is no distinct sulcus between the lid itself and the fold. In the case described by von Graefe a piece of cartilage was found in the base of the fold, and in that described by Oeller there was a large defect in the cartilage and the lid coverings. Most of the cases have been found in Jews. From the data it appears that many more of the milder types occur than are reported, for Schapringer commented on the number that he found after he had seen the first few cases. The first case in the literature was described by Blasius in 1835, and that reported by von Graefe is of especial interest. It did not receive special study until 1899, when Schapringer of the German Hospital clinic in New York reported eight cases. This number was soon augmented. Wibaut has found the greatest number, and Fuchs mentioned the condition as a sequel of croupous conjunctivitis, the inflammation producing the adhe-

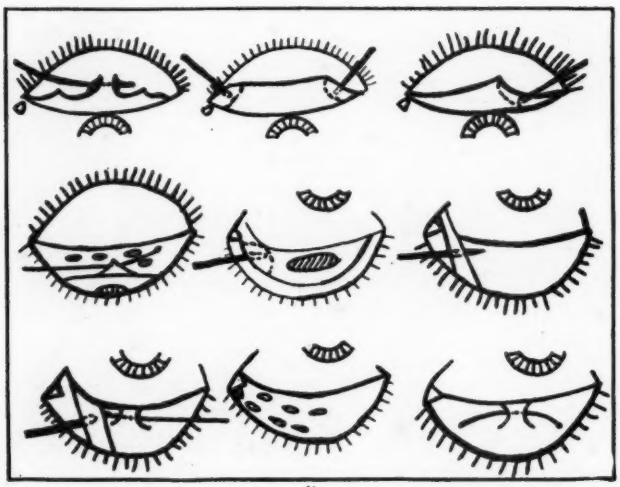

Fig. 3.—Types of epitarsus (Wibaut).

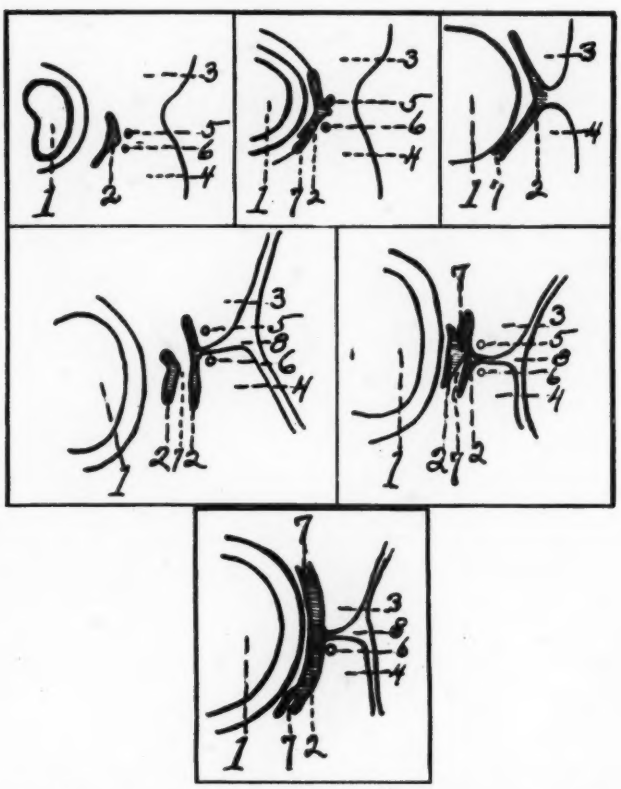

Fig. 4.—Development of the conjunctival sac and the plica semilunaris (Wibaut): *1*, bulbus; *2*, conjunctival sac; *3*, upper lid; *4*, lower lid; *5* and *6*, upper and lower canaliculi; *7*, anlage of the plica semilunaris; *8*, epithelial adhesion of the lids. Sections made through the inner angle of the lids, vertically. The first three sections are from an embryo of 35 mm., and the second three sections from one of 40 mm.

sion of the hypertrophied fold which is assumed to drop down from the fornix because the tissues are so swollen and thickened. That the traction of such a band could cause inversion of the lid in a new-born infant does not seem possible. If the present case is to be used as a standard, it must be said that any great development of such an anomaly implies associated errors of development of the tarsal cartilage which complicate the corrective operative procedure and modify the prognosis. Some of the cases reported were so simple that it was easy to snip off the narrow and attenuated basal attachment in the fornix, but this patient will probably always have peculiar upper lids, because they lack the shape and firmness for which a normal tarsal cartilage is responsible. Whether this child will ever be able to raise the upper lids is a question, for there has been no indication of this power. As the upper margins of the tarsal cartilage were everted, it is difficult to imagine a normal connection of this tissue with the tendinous expansion of the levator palpebrae superioris.

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Ophthalmologic Review

RETINAL CHANGES IN HYPERTENSION AND IN RENAL DISEASE

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More than seven decades have passed since the discovery of retinal changes in Bright's disease. In this interval a belief in their ominous prognostic significance has become established, and for this reason many internists have learned to use the ophthalmoscope. A great deal of experience has been gained, and much knowledge has been obtained, but ignorance on some important points remains.

It is rapidly being accepted that the retinal changes do not occur in renal disease unless hypertension is present, no matter how gravely the function and structure of the kidney is damaged.

Decided changes in the eyeground unquestionably mean danger to life, but it has become possible and important to place very different prognostic values on the different appearances of the fundus.

Necessary to the understanding of this expanding problem is some knowledge of the changes in the eyeground during life, of the retinal lesions after death, and of the present conception of renal disease and hypertension. These will be presented in brief review, and then, with these data as a basis, the relations of retinal changes to hypertension and renal disease will be outlined.

CHANGES IN THE EYEGROUND

The changes in the eyeground comprise changes in the vessels and changes in the retina itself. Some of the changes in the vessels are functional and temporary, while others are organic and permanent.

Many observers have described a narrowing of the arteries, especially of the smaller ones. This narrowing, or attenuation, has been seen not only to appear and disappear but at times to affect only portions of the vessels and at other times the whole length. These observations have been confirmed by retinal photographs. This appearance, then, can properly be ascribed to a spastic contraction of the media of the vessels concerned. It occurs sometimes as the only change, but is frequently associated with sclerosis of the retinal vessels and is seen in fundi that show hemorrhages, white patches and swelling of the nerve head. In gravid women its increase and extension have been studied, and white patches and hemorrhages have been seen to appear when the spasm reached its fullest development. This observation gives support to the

theory that "retinitis" is caused by ischemia. Another functional change is dilatation of the veins. This is especially associated with choked disk, but also appears in sclerosis and retinitis.

Sclerosis of the retinal arteries is most distinctly evidenced by pronounced and sudden alterations in the caliber of an artery. Other changes that indicate sclerosis are a loss of translucency, an increased brilliance of the central light streak, a whitish line along either side of the artery and a copper color. An increase in length, even to corkscrew tortuosity, often occurs in sclerotic arteries both in the eyes and elsewhere, but it is not an essential characteristic. Compression of the larger venous branches where the arteries cross them is regarded as important. Competent observers believe that the vein is pushed into the retina rather than compressed. Miliary aneurysms are rare; multiple ones are described. White threads that result from arterial thrombosis and obliteration are occasionally seen. The venous walls also may be thickened. Retinal sclerosis may occur alone or in fundi that show hemorrhages, white patches and a swollen disk. Sclerosis is not, however, a necessary concomitant of neuroretinitis.

Hemorrhages into the retina vary in number and in size. They may be clearly outlined when superficial or more undefined when deep. Small ones are often linear and parallel to the vessels. They are apt to be unequal in the two eyes and not infrequently occur only in one. They often occur with arterial spasm and with sclerosis, and are usually present in fundi that show white patches.

"White" patches that vary in tint from white to yellow and in area from punctate to disk size have long been an important component of the changes in the fundus in renal disease. They are usually situated near the posterior pole and especially between the disk and the macula. Recently great emphasis has been placed on whether the margins of these patches are cleancut ("hard" patches) or hazy ("cotton wool" patches). No sharp distinction can be made between some large hazy patches, especially if confluent, and large ill defined areas of lighter color in the retina, the so-called "zones of edema." The stellate figure about the macula, usually most perfect on the nasal side, is made up of small, sharply defined patches.

The sharply outlined patches often occur with hemorrhages and arterial change, but with nothing else. On the other hand, they are regularly present when "cotton wool" patches and blurred disks combine to produce neuroretinitis.

Elevation of the disk with blurring of its margins is not unusual. The development of this condition varies greatly. It may occur alone, when it is called "choked disk," and it may be unilateral. The swollen disk may be redder or paler than normal owing to congestion or ischemia. Anemia may help to blanch it. In long-standing cases, the disk

may become atrophic and white. Swollen disks may be associated with "cotton wool" or "hard" patches and hemorrhages in the retina, and the entire lesion is then called "neuroretinitis." In such fundi, the veins are regularly dilated. Spasm of the arteries is usually present, but sclerosis not so regularly.

RETINAL LESIONS

Retinal lesions have been studied chiefly in the eyes of people who have died of nephritis. It is assumed that the retinal lesions in essential hypertension are the same as those in nephritis when the appearances of the fundus in life are the same. This assumption is further supported by an insufficient but increasing number of autopsies.

In general, microscopic examination after death shows the changes in retinal structure to be much more extensive than did ophthalmoscopic examination during life.

The edema that occurs in the retina and in the nerve head does not show any peculiar characteristics. When the edema is decided, the internal limiting membrane may be thrown into folds. An effusion of fluid may occur between the retina and the choroid, lifting the former. This is believed to be an agonal event in many instances, but not in all. In long-standing cases, atrophy of the nerve head may be found.

Hemorrhages varying in size occur in all layers of the retina, especially in the nerve fiber and internuclear layers. They also may occur between the retina and the choroid. The blood is believed to escape chiefly from the capillaries.

Sclerosis of the retinal arteries is frequent, but it is no longer considered to be present necessarily when lesions in the retina itself occur.

Various descriptions are given of the arterial lesions, but Schieck is the authority for the statement that there is nothing to distinguish them from those of sclerosis elsewhere in the body. "Obliterating endarteritis," which Volhard believes to be a typical result of ischemia, also occurs.

Degeneration of the retinal tissue itself takes different forms. First, in the nerve fiber layer there are lesions which consist of peculiar local varicosities of a group of nerve fibers. Edema develops about them, raising the internal limiting membrane. The swollen fibers ultimately disintegrate, and fat or lipoid is deposited in the homogeneous or granular mass that remains.

There are also deeper lesions in the outer plexiform layer invading both juxtaposed nuclear layers. Some are simply zones in which the retinal tissue contains a considerable deposit of yellow amorphous substance, fat or lipoid. When such an amount of fat occurs within the retinal elements, the lesion is looked on as retinal degeneration rather than infiltration. There is no destruction of retinal elements in these zones. Next there are zones in which the retinal elements are destroyed,

and in their place there is a tangled mass of fibrils suggesting fibrin. These fibrils, however, do not take the fibrin stain, and are continuous with retinal structures at the margin of the lesions. Considerable fat occurs in these lesions. Finally, there are sharply limited rounded zones in which all retinal structure is lost. These zones constitute small cavities usually filled with a dense coagulum that shows traces of fibrillar structure after hardening, "Faserkorbe," or fiber baskets. They contain much fat and some fibrin. Sometimes the coagulum is much less dense and contains some red cells and some other cells. Large phagocytes filled with fat are often present in all the destructive lesions. It cannot be determined by the ophthalmoscope during life which lesion causes a particular patch. A white or yellow patch can probably be produced by local edema, by an area of fat deposit, by varicose nerve fibers, by a reticular zone, by a "fiber basket," by fibrin or by altering blood pigment in an absorbing hemorrhage. It is also possible that subretinal transudates may shine through as areas whiter than the surrounding retina.

The reason the patches are disposed in a stellar figure about the macula is still under discussion. Some observers believe that this depends on the radial disposition of the blood vessels in this region; others, that it depends on a similar radial arrangement of the Müller fibers. As rarities, pigmentation of the retina sequent to nephritic retinitis and areas of calcification have been described.

The pathogenesis of the foregoing lesions is so intimately bound up with their significance in hypertension and nephritis that discussion of it is deferred till the current views on renal disease and high blood pressure have been outlined.

RENAL DISEASE AND HYPERTENSION

The conception of the cases grouped under the head of Bright's disease has been undergoing expansion and elaboration ever since Bright established the coincidence of dropsy, albuminuria and gross lesions in the kidneys a century ago.

Nitrogenous waste is formed from body tissue no matter to what extent nitrogenous food is restricted. The kidney is the only organ that can remove this waste from the body fast enough to prevent its retention. When nitrogenous waste accumulates in the body, it poisons the system and finally proves fatal. Life thus depends on adequate renal function. It has long been known that more blood flows to the kidneys in proportion to their weight than to any other organ. The necessity for respiration is so immediate that a separate circulation has been evolved to insure the passage of the entire volume of blood through the lungs. The necessity for purging the blood of nitrogen is not so immediate, but ultimately it is no less vital. A large proportion of the blood, then, must pass through the kidney. No such necessity obtains

in regard to any other organ in the systemic circulation. This is presumably the explanation of the proportionately huge volume shunted to the kidneys. It is reasonable to suppose that if the flow of blood to the kidneys becomes difficult the flow to the rest of the body must be made correspondingly difficult, through spastic narrowing of the systemic arterioles, in order to insure to the kidneys the necessary proportion of blood. This is one explanation of the genesis of hypertension, but there is an altogether opposite view, which will be presented later.

There are four main components of renal tissue: the glomeruli, the uriniferous tubules, the blood vessels and the supporting stroma. When kidneys from people who have had albuminuria, dropsy, hypertension and retention of nitrogen are examined after death, a great variety of lesions are found. It is believed that lesions of the tubules may cause defective excretion, that lesions of the glomeruli may cause both defective excretion and elevation of blood pressure, and that lesions of the arteries may cause elevation of blood pressure. It is not believed that lesions of the stroma directly affect either renal excretion or blood pressure. At any rate, there are extensive lesions of the secretory tubes and glomeruli in most persons who die of nitrogenous retention and lesions of the arteries in most of those who die of hypertension.

On this basis, the modern classification divides renal lesions into three main groups: those of the tubular epithelium, those of the glomeruli and those of the arteries. The attempt is then made to ascribe distinctive symptoms to each of these groups of lesions. These proposed clinicopathologic groups have been called nephrosis, glomerulonephritis and renal sclerosis, respectively. The nephrosis group may quickly be disposed of, because in it hypertension is most unusual and changes in the eyeground have not been described, except in the presence of amyloid kidneys; the latter condition has been included in this group under some protest. Hypertension may develop in cases of advanced amyloid lesions, and as a great rarity, hemorrhages and "hard" patches have been observed. Fishberg and Oppenheimer reported such a case.

In glomerulonephritis, the early changes concern chiefly the glomeruli, and some hypertension develops at the onset before any changes in the arteries have occurred. As this lesion advances, the tubes are increasingly damaged, the stroma grows, and sclerosis of the arteries develops, often to a marked degree. With progress of the lesion, the blood pressure continues to rise, and renal failure develops. The final stage has been called secondary atrophy (secondary to an inflammation of the kidney). Neuroretinitis is frequently present before death.

Renal sclerosis includes all the lesions in which sclerosis of the renal arteries is believed to be the primary lesion. There are cases in which the only change is sclerosis of the arteries. Hypertension is present in the majority of these. Then there are a large number of cases in

which zones of secretory tissue (tubes and glomeruli) are throttled by an overgrowth of stroma, in addition to the sclerosed vessels. Such lesions are considered ischemic by most modern observers. The secretory tissue outside the zones of destruction remains comparatively normal, and renal function remains undisturbed. Hypertension is usually associated with such lesions. In all the foregoing cases, the condition has been called "benign" sclerosis because renal failure does not occur. Finally, there are cases with advanced sclerosis, stenosis and sometimes necrosis of the arteries, extensive changes in the secretory tissue and massive overgrowth of stroma. The secretory tissue outside the zones of stroma growth is damaged. In these cases death is due to combined renal and cardiac failure. The condition in these cases has been called "malignant" sclerosis, because renal failure impends, and "primary" atrophy because the disease does not originate in acute nephritis. Neuroretinitis is usually, but by no means inevitably, present in the eyes. In general, "malignant" sclerosis appears earlier in life than "benign."

This classification, when applied to renal cases, leaves much to be desired. The lesions are rarely of pure type. At necropsy, many kidneys show lesions so mixed that it is impossible to say which component is primary, dominant and essential. Widely divergent views are held as to their genesis. The distinction between "primary" and "secondary" atrophy is especially difficult.

As to symptoms, there is not one that is constantly present in any one of the foregoing groups and that is always absent when only lesions of other groups exist. Confusion in making the clinical diagnosis of the lesion is an ordinary occurrence.

A more detailed discussion of this enormous problem will lead too far from the subject under consideration, but it must be clearly understood that the foregoing classification of Bright's disease is far from being adequate or complete.

Hypertension, the symptom on which changes in the fundus primarily depend, is a good illustration of the difficulties of referring a symptom to a lesion in Bright's disease. In nearly all cases of hypertension of long standing, sclerosis of the renal arteries is found at death, and probably a majority of people who have sclerosis of their smaller renal arteries have hypertension during life. But gravid women, some persons with suprarenal tumors, those with acute glomerulonephritis and some with normal kidneys have pronounced hypertension without renal sclerosis, while sclerosis of the renal arteries is found in a number of people who have never had hypertension. Moreover, there is diametrical disagreement about the relation of hypertension to sclerosis, some observers believing, as stated previously, that renal sclerosis induces hypertension, others that general hypertension is the

primary event, and that sclerosis of the small arteries, not only in the kidney but elsewhere in the body, is one of its results. Still others believe that renal sclerosis may cause hypertension in some instances, and that hypertension may cause sclerosis of smaller arteries in others. Another discussion exists as to the mechanism by which the arterioles are stimulated to spastic contraction. Volhard expressed the belief that a less dangerous form of tension is produced by the vasomotor center because the distribution of blood to the different organs remains proportionate to their needs, and that a more dangerous form is caused by a chemical stimulus in the blood which affects all vessels alike and so deprives vital organs, as well as less important ones, of their necessary supply. Kahler and others maintain, on the contrary, that the stimulus that causes all the important and serious forms of arteriolar spasm emanates from the vasomotor center in the medulla. Then there are some observers who believe that an excess of sodium chloride in the body causes hypertension, and that the pressure may be reduced by limiting salt in the food. But there are more observers who deny that salt exerts any significant influence on blood pressure. Hypertension constitutes the prerequisite condition for the development of the retinal lesions here described, but knowledge of more detailed relationships between the kind of tension and the kind of retinal lesion must wait till the various problems concerning the genesis of hypertension itself approach solution.

PATHOGENESIS OF THE RETINAL LESIONS

In general, all the changes in the retina and its vessels, so far as they are incident to hypertension and renal disease, are primarily due to the hypertension; without this they do not occur. Grave destruction of the kidneys as seen not only in Bright's disease but also in renal tuberculosis, septic kidneys and renal tumors, does not cause any abnormality of the fundi unless hypertension develops. Neither hypertension nor retinitis is usually found in children with glomerulonephritis nor in persons with glomerulonephritis as a complication of bacterial endocarditis. Similarly, changes in the retinal tissue itself are not seen in cases of advanced arteriosclerosis without hypertension.

Sclerosis of the retinal vessels, then, may develop in people who have never had hypertension and who have sclerosis of the arteries in various other parts of their bodies; but in people who have had hypertension for a few years, retinal sclerosis is nearly inevitable. Therefore, there is much reason to believe that hypertension may produce retinal sclerosis.

Hemorrhages in the retina occur in many conditions, such as anemia, in which hypertension has never existed. On the other hand, they are frequent in severe hypertension and are rarely seen in renal disease unless the blood pressure is or has been high.

So far as the degenerations of the retina are concerned, hypertension is regarded as an essential prerequisite to their existence in nephritis. It is further believed by many that it is through ischemia that hypertension causes degenerations, the spasm narrowing the lumina of the retinal arterioles to such an extent that the increase of pressure in the arterial tree is insufficient to drive the normal or necessary amount of blood through their reduced bore.

In gravid women, the small retinal arteries have been seen to grow progressively narrower, and when this change has reached its maximum white patches have appeared.

The original idea that retinitis was related in some definite way to albumin in the urine, which gave rise to the term "albuminuric" retinitis, has been entirely discarded.

Retinal arteriosclerosis was formerly thought to be the main cause of retinitis, but its independent occurrence makes this theory untenable. That sclerotic stenosis may aggravate the ischemia cannot be doubted.

Widal thought retained nitrogenous waste was a chief cause and spoke of "azotemic" retinitis. But large and fatal retention of nitrogen occurs so often without affecting the fundi, unless hypertension coexists, that this view also has been abandoned. There is, however, reason to believe that retention of nitrogen or some toxin derived from the failing kidney is a contributory factor in neuroretinitis.

Chauffard found lipoid in the retinal lesions and an increased percentage of cholesterol in the blood, and suggested that cholesterolemia might prove to be a necessary condition in the genesis of retinitis. Again there are too many exceptions. The highest amount of cholesterol in the blood in renal disease occurs in lipoid nephrosis in which retinitis is practically unknown, and a wealth of retinal patches may occur when the blood fat is normal. That a high amount of cholesterol in the blood favors the development of patches and that damaged retinal tissue may invite deposits of lipoid even when the blood fat is not above normal, seem to be the most recent views in this matter.

The genesis of white patches in the retina, then, depends primarily on hypertension, and this is regarded as an ischemic effect through spastic reduction of the bore of the arterioles. Arteriosclerosis surely may aggravate the ischemia, but unless hypertension is present it does not produce the lesion so far as observed. A high amount of cholesterol in the blood is probably a contributory factor. That a toxic factor cooperates with hypertension in the production of severe lesions is a widely held belief.

Swelling of the nerve head due to congestion or edema or both is of dual origin, and, unlike the zones of retinal degeneration, is not dependent on hypertension alone. It occurs when hypertension and renal failure are present, and then forms an essential part of the lesion called

neuroretinitis. It is stated by Fishberg and Oppenheimer frequently to be the first element of this lesion to appear. Whether in these cases it is an ischemic or a toxic effect is, so far as I have learned, undetermined. Papilledema may also develop in the absence of hypertension due to an increase of intracranial pressure incident to edema, tumor of the brain, etc. It is usually the only change and is called "choked disk." Hemorrhages in the retina may be associated with it, and even patches of retinal degeneration may occur with this type of papilledema. This is believed to be due to pressure through interference with venous or lymph flow, because the swelling regularly disappears when the pressure is relieved.

CHANGES IN THE FUNDUS IN HYPERTENSION AND IN RENAL DISEASE

Narrowing of the retinal arteries alone has been observed chiefly in gravid women and in persons with acute glomerulonephritis. It occurs in marked development associated with sclerosis, hemorrhages, patches and swollen disks in late chronic nephritis and in the malignant form of sclerosis.

Sclerosis of the retinal arteries alone is present almost constantly in people who have suffered from hypertension for a few years. It also may be present as part of a general arteriosclerosis in people whose blood pressure has never been elevated. It occurs with hemorrhages and sharply defined white patches in hypertension when the renal function is normal. This not infrequent combination has been called "arteriosclerotic retinopathy." Sclerosis is frequently but not constantly present in fully developed neuroretinitis.

Hemorrhages in cases of hypertension do not usually occur unless some sclerosis coexists. They are often associated with this alone, and are often coincident with patches of sharp outline only. They form a regular component of neuroretinitis, and may occur also with choked disk when hypertension has never been present.

The whitish patches are the changes in the fundus about which most of the present confusion obtains, partly because it is yet impossible to say from looking at them just what retinal lesion is present and partly because great clinical importance is attached to the sharpness of their margins, a feature that cannot be satisfactorily studied in the retina after death. The "hard" patches not infrequently occur with hemorrhages and sclerosis, in the absence of "cotton wool" patches and papilledema, in cases of hypertension without renal failure, the so-called "arteriosclerotic retinopathy." They also form a constant component of typical nephritic neuroretinitis. The macular star, when present, is made up of a number of these "hard" patches, radially disposed. The "cotton wool" patches combine with papilledema to form the essential features of nephritic neuroretinitis. "Hard" patches and hemorrhages regularly coexist, but are not essential to this condition. Sclerosed arteries,

dilated veins and spastic arterioles are also often present, but they are not essential. "Cotton wool" patches rarely occur unless there is some alteration of the nerve head.

Neuroretinitis occurs chiefly in malignant sclerosis and in the final decompensated stages of chronic glomerulonephritis, that is, in "primary" and "secondary" renal atrophy, "genuine" and "secundäre Schrumpfniere." In these cases, renal failure is added to hypertension. Typical neuroretinitis may occur and persist for years in cases of hypertension, and renal function remain normal throughout the entire period. On the other hand, in cases of hypertension, death due to renal disease not infrequently occurs without any changes in the retinal tissue.

Swelling of the nerve head and blurring of its margins are the most essential characteristics of neuroretinitis, the clinical incidence of which has just been stated.

When papilledema occurs alone or associated only with retinal hemorrhages, it may be the precursor of neuroretinitis or it may be due to increased intracranial pressure, in the absence of hypertension. It is also possible, though exceptional, for the whole picture of nephritic neuroretinitis to be produced by intracranial pressure.

DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE OF CHANGES IN THE FUNDUS

Narrowing of the retinal vessels means spasm, and indicates that blood pressure is elevated. It cannot have any further significance by itself. Sclerosis of the retinal arteries alone usually means that hypertension has existed for some time; it also indicates that arteries elsewhere are similarly affected. Sclerosis may exist without hypertension, and hypertension without sclerosis. It is not thought to be evidence of renal disease. Many observers believe that apoplexy occurs more frequently in people whose retinal arteries are sclerosed than it does in people who have sclerotic vessels elsewhere but none in the retina.

Changes in the retina itself have long been thought to mean impending renal death within two years. This tenet has got to be changed and enlarged. Hemorrhages and sharply defined white patches often persist for years in cases of hypertension in which renal failure never develops. It is probably safe to conclude that these changes do not indicate any special liability to death from renal disease. On the other hand, persons with hypertension with these changes in the fundi are in general more liable to die from cardiac or cerebral conditions than those in whom these changes are absent. There are notable exceptions in which these changes have failed to progress over several years, and others in which they have receded. They certainly do not place a definite limit on life. On the other hand, hemorrhages and patches do

not carry any guarantee that renal failure is absent or that it will not occur any more than normal fundi do. It is only their presence and not their absence that is significant.

"Cotton wool" patches and papilledema combine to form nephritic neuroretinitis. Changes in the vessels, hemorrhages, "hard" patches and a macular star usually coexist. This lesion is called "typical nephritic retinitis" by Kahler and Sallmann; "malignant hypertensive neuroretinitis" by Fishberg and Oppenheimer and "retinitis of malignant hypertension" by Keith, Wagener and Kernochan. Such a fundus means, in general, that the person who has it will die within two years. Death is usually uremic, but may be due to apoplexy, cardiac failure or intercurrent disease. Such a retinitis not infrequently appears some time before renal failure supervenes. Keith, Wagener and Kernochan seem to regard it as tantamount to a death sentence. This view, though generally held, is not quite justified. Fishberg and Oppenheimer described such a retinitis in a case of suprarenal tumor in which renal function was normal; it has similarly been seen in a case of lead poisoning, and Kahler and Sallmann, on the basis of an extensive experience, reported eight cases observed over long periods in which this change in the eyeground was present but in which, so far as is known, renal failure never developed. It is further remarkable that such eyegrounds may regain a normal appearance. Kahler and Sallmann reported such cases; Schieck called attention to similar complete recessions, and one instance occurred in my own experience.

How "cotton wool" patches and a swollen nerve head are produced in anticipation of renal failure is not known. Fishberg and Oppenheimer found necrosis of the renal arterioles at autopsy in every one of ten cases of nephritic neuroretinitis in "malignant sclerosis" ("primary" atrophy). In one case of chronic nephritis ("secondary" atrophy) that came to autopsy this necrosis was not found.

The fundus of "arteriosclerotic retinopathy" and that of "nephritic neuroretinitis" do not stand as two sharply distinguished types, for many eyegrounds present pictures intermediate between them, and their significance becomes correspondingly uncertain.

Papilledema occurring alone may be the first indication of nephritic retinitis, or it may indicate increased intracranial pressure either from an edematous condition or from tumor of the brain. It is significant of danger, but just what that danger is requires further observation to determine. The exceptional cases in which intracranial pressure produces the complete picture of nephritic neuroretinitis must always be borne in mind.

In the present state of knowledge, then, retinal sclerosis calls for a study of the blood pressure; hemorrhages and "hard" patches in the retina bespeak a serious type of hypertension with circulatory danger, but do not set a definite limit to life; nephritic neuroretinitis means, in

the vast majority of cases, renal or circulatory death within two years, though surprising exceptions occur; papilledema alone is ominous, but requires observation for its interpretation. The absence of changes in the retinal tissue does not exclude grave hypertension or advancing renal failure.

Until renal lesions can be diagnosed with greater certainty during life, misleading statements will be avoided at the bedside by speaking of the relation of various changes in the fundus to disturbances of function, i. e., to hypertension and to renal failure rather than of their relation to particular renal lesions.

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News and Notes

GENERAL NEWS

Chicago Graduate Course in Ophthalmology.—Owing to general conditions, it has been deemed advisable to postpone the Chicago Graduate Course in Ophthalmology for one year. The present plans call for the beginning of the course in September, 1932. Applications will be received by the Secretary, Dr. R. C. Gamble, 30 North Michigan Avenue, Chicago, up to July 15, 1932.

Blind People Affected by City Noise.—A survey, made by the New York Association for the Blind, has revealed that the effect of the city's noise on blind people makes it practically impossible for them to walk in a straight line. The noise drowns the sound of the blind person's footsteps, by which he is ordinarily guided, and the sound of the feet of other pedestrians. This survey was conducted by interviewing a large number of blind people of all types, and by calling on the experience of workers with the blind.

Course for Ophthalmic Residents.—The course in ophthalmology for ophthalmic residents in Chicago will be given by members of the schools and hospitals having such residents from September to December, as was done last year.

PERSONAL

Dr. Nikolaus Blatt, of Targu-Mures, Roumania, has been appointed Privat Dozent on the medical faculty of the University of Bucharest in ophthalmology. His first courses will be held from October to November, 1931. The department is in charge of Professor Manolescu.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE DEVELOPMENT OF THE VITREOUS BODY IN THE FOWL. E. REDSLOB, Ann. d'ocul. **168:** 29 (Jan.) 1931.

In the fowl the vitreous develops from a basal membrane, which originates exclusively from the ectoderm. It seems to be produced by an intercellular substance which divides, accumulates and condenses on the border of the ectodermal layer. In the interior of the cavity, the basal membrane resolves itself into a veil of amorphous substance, which represents the first stage in the vitreous formation. The intercellular substance, the basal membrane and the amorphous veil all take the color of gelatin. This does not mean that these three elements are gelatinous.

All the solid parts of the vitreous originate from a basal membrane which, at a certain time, detaches itself from the epithelial layer and penetrates alone into the vitreous cavity where it continues to resolve itself into an amorphous substance, and disappears. A basal membrane then forms itself as a border around the retinal or crystalline epithelium. When these have been exhausted of their material, they form into others and so on until finally all the basal membranes disappear except the one that is left at the level of the internal border of the retina where it later develops the ciliary body. This repetition is continued for a long period with distribution of its amorphous substance into the interior of the cavity.

The most important phenomenon, and the one that shows best the mechanism of the vitreous formation, is produced at the level of the embryonic furrow, the external basal membrane of the retina which is separated by retinal sheaths for the vitreous at the level of the embryonic furrow, its epithelial support crossing the furrow and reaching the cavity in which it penetrates deeply. Here it throws off material which is shed about. It is a curious fact that a basal membrane so far away participates in the formation of the vitreous.

At no time does one find mesodermic elements in any form in the interior of the vitreous cavity. At no time does the mesoderm take part in the vitreous formation. When one has seen in the formation of the vitreous body that the accumulation of the amorphous substance takes place by successive processes, one can understand the formation of the different strata in the living vitreous visible by the slit-lamp.

S. HANFORD MCKEE.

THE DEVELOPMENT OF THE ANTERIOR CHAMBER AND OF THE CORNEAL ENDOTHELIUM. R. SONDERMANN, Arch. f. Ophth. **125:** 407, 1930.

In contradiction to the generally accepted view, the author believes that the endothelium of the cornea is of ectodermal origin. The thick layer of ectoderm that lies in front of the lens vesicle is invaded by scleral fibers on each side so as to form an ectodermal lining of those fibers. The anterior chamber is formed in intimate connection with the

development of the pupillary membrane, the capillaries of which produce the first aqueous by filtration. This process of filtration is started by the increasing resistance that the vena iridoscleralis encounters on its way through the sclera. In a small percentage of cases the formation of the anterior chamber may be delayed for several months.

P. C. KRONFELD.

Bacteriology and Serology

AUTOSEROTHERAPY IN TRACHOMA. N. FEHMI, Rev. internat. du trachome 8: 68 (April) 1931.

The biologic researches of Kleczowski and Karelus have brought forth the fact that a certain toxic element exists in the serum of the trachomatous patient. The work of Sgrosso on this subject has opened a vast horizon of hope in the treatment for trachoma. Augelucci, and more recently his pupil Salvati, have introduced the method of autoserotherapy in trachoma. The author has tried this method in fifty cases of trachoma of various types and stages. Twenty were acute cases, sixteen were in the cicatricial stage and fourteen were complicated by corneal ulcerations and pannus. Thirty cubic centimeters of blood was taken and the serum separated out was inactivated at 56 C. for one-half hour. Ampules of 0.5 cc. each were then prepared. The injections were given every two hours under the palpebral conjunctiva of the upper lid. Comparisons were made by treating one eye only, while the other eye received the usual silver nitrate, copper and grattage. During the treatment, which lasted two months, each person received from 15 to 20 cc. of serum. The eye receiving this kind of treatment had no additional care. The results were as follows:

1. There was no local reaction at the point of injection in the upper lid.

2. There was no sign of general reaction early or late—no pain, fever or headache.

3. In the acute trachomas there was a diminution of the congestion, lacrimation and photophobia after six or seven injections.

4. In the complicated cases there was no change; the ulcers and pannus were not affected.

5. The conjunctival hypertrophy was not influenced, while the eyes given the classic treatment showed notable improvement.

These were true cases of trachoma. The serums were prepared by Dr. Chukri, a serologist. The author does not think that autoserum is an ideal treatment for trachoma.

L. L. MAYER.

EXAMINATION AND TREATMENT OF THE APPARENTLY NORMAL CONJUNCTIVA PRIOR TO OPERATIONS ON THE GLOBE. W. JAHNKE, Klin. Monatsbl. f. Augenh. 85: 644 (Nov. 28) 1930.

The opinion of operators differs regarding the danger of infection in the apparently normal conjunctiva in operations that require an incision into the globe. Climatic conditions, the stage of civilization of the population and the skill of the operator are of consequence in the prevention or development of these postoperative infections. Jahnke reports

his experiences at Krueckmann's University Eye Clinic at Berlin, Germany. The number and virulence of the germs in the conjunctival sac were of great importance. Virulent germs were found in the conjunctival sac of 43 per cent of the patients prior to the operation. Streptopneumococci and certain forms of staphylococci were found. A hemolytic and a nonhemolytic form of Staphylococcus aureus and hemolytic Staph. albus were found, besides the hemolytic and the nonhemolytic streptococci, usually of the viridans type.

The best results were obtained with Schottmüller's blood agar mixture, consisting of 2 cc. of human blood to 5 cc. of a 2 per cent solution of agar on a petri dish. Differentiation in mixed infections and the degree of hemolysis can be well studied (Schottmüller, Hugo: Leitfaden für klinisch-bakteriologischen Kulturmethoden, Berlin, Urban & Schwarzenberg. 1923). Ninety per cent of the pneumococci belonged to the nonagglutinable type, which, incidentally, were found to be the main cause of serpent ulcer; these were least influenced by serum therapy.

Sterilization of the conjunctival sac was obtained by Betti's method, the application of a 2 per cent salve of yellow oxide of mercury and a bandage. The bandage was changed once a day for three days, after which a smear and a culture were made. Another bandage was applied in the same manner and was left in place until shortly before the operation if, after twenty-four hours, both the smear and the culture proved to be sterile. The yellow ointment, prepared after Schweissinger's method, was equally as effective as that of Betti, which has the following formula: To 0.2 Gm. of yellow mercuric oxide, add 1 cc. of distilled water; mix well and add 2 Gm. of hydrous wool fat, liquefied, in a double boiler; mix again and add 8 Gm. of purest petrolatum; mix very well.

The hyperemic conjunctiva of patients suffering from glaucoma, who had been treated with pilocarpine over an extended period, was usually found free from germs. Nevertheless, the patients received Betti's treatment prior to the operation.

K. L. STOLL.

Conjunctiva

AN EPIDEMIC OF CONJUNCTIVITIS DUE TO INFECTION WITH BACILLUS FAECALIS-ALKALIGENES. R. H. B. BARROW, Brit. J. Ophth. **15:** 319 (June) 1931.

The outbreak occurred in a training ship for boys for the Royal Navy and the Merchant Service. The ship lies at anchor in the Thames. The boys with the original cases had recently joined the ship and had evidence of old trachoma. Notwithstanding isolation of the first nine boys affected, there was a rapid spread of the infection. There was a general hyperemia of both bulbar and palpebral conjunctiva with follicles present in the upper fornix along the tarsal margin. The lower fornix was free from follicles except in one instance. There was a small amount of mucopurulent discharge. The condition was similar to the early stage of trachoma.

The causative organism was a gram-negative, motile bacillus, a short rod from 0.5 to 1 to 2 microns long, occurring singly or in pairs, occa-

sionally in long chains, and provided with peritrichous flagella. Petruschky found it originally in spoiled beer, and it has been found frequently in water. A study of the effects of various solutions used led to the conclusion that washing out the conjunctival sac is the essential, and that probably physiologic solution of sodium chloride would have been equally efficacious.

W. ZENTMAYER.

CONJUNCTIVAL AND EPISCLERAL VASODILATATION AFTER MUMPS. A. COLRAT, Arch. d'opht. 47: 839 (Dec.) 1930.

The rarity of ocular complications with mumps prompts the author to report briefly a case in which the conjunctival and episcleral vessels became dilated seven days after the onset. There were no other ocular lesions found. There was no swelling of the lacrimal glands, no secretion from the conjunctiva, no evidence of iritis. The author was unable to find a similar case in the literature. The symptoms subsided at the end of two weeks.

S. B. MARLOW.

NEW OPERATIVE PROCEDURE FOR PTERYGIUM. GOMEZ MARQUEZ, Arch. de oftal. hispano-am. 31: 87 (Feb.) 1931.

The author is inclined to consider pterygium a neoplasm. He details his operation, which consists essentially in a radical extirpation of the growth and a covering of the defect with a conjunctival flap taken from the upper scleral conjunctiva of the other eye and held in place with sutures.

C. E. FINLAY.

PSEUDOTRACHOMATOUS CONJUNCTIVITIS WITH DIFFUSE TARSITIS OF SYPHILITIC ORIGIN. BURCUS, Cluj. med. 11: 418, 1930.

A young man, 20 years old, presented follicles on the superior and inferior tarsus of the left eye and an extensive pannus. The condition remained unaltered in spite of antitrachomatous treatment conducted for two months. The preauricular lymph nodes were markedly enlarged. After three months there were follicles and scars on the conjunctiva, pannus and a decrease in the acuity of vision to perception of light. The history disclosed a syphilitic infection acquired three months prior to the appearance of the disease of the eye. A strongly positive Wassermann reaction was present. After the first injections of mercury and arsphenamine the condition of the eye improved, the pannus receded, and the acuity of vision increased. After three courses of antisyphilitic injections the pannus disappeared completely, and vision was 1/6.

N. BLATT.

Cornea and Sclera

SUCCESSFUL GRAFTING OF CORNEA IN RABBITS. J. W. T. THOMAS, Lancet 1: 335 (Feb. 14) 1931.

From experimental work covering eight and a half years, Thomas concludes that transplantation of the cornea in rabbits can be successfully carried out. If corneal transplantation is to be applied to man in order to remedy blindness, it is reasonable to assume that methods which have

proved inefficient on normal eyes would not be successful when operating on a cornea damaged as a result of previous disease or injury. The author points out, however, that the cornea of a rabbit is thinner than that of man, so that apposition of the margins of the graft has to be all the more accurate. Furthermore, it is more difficult to assure asepsis in rabbits during the period of healing, as the animal is not under the same control as a patient lying in bed. In this way, slight defects in the healing of the graft are apt to lead to more serious consequences; if, for example, the edge of the graft at one spot overlaps the adjacent cornea, it affords an excellent opportunity for the entrance of bacteria, and septic complications, which are so damaging in their effects, are likely to result. Of the six methods of corneal grafting which he described, one appears to offer much better chances of success than do the others, so that of the seven rabbits operated on by this method, all of the grafts united, five were transparent, one had a central clear area, and only one was opaque.

W. ZENTMAYER.

SYMMETRICAL CENTRAL LIPOID INFILTRATION OF BOTH CORNEAS IN A DOG. M. DREYFUSS, Arch. f. Ophth. **125**: 67, 1930.

In the right eye of a German shepherd dog, 10 years of age, was found a deep, round corneal opacity, two millimeters in diameter, while in the left eye were two similar, but smaller, lesions. The eyes had never been inflamed or injured. The opacities were yellow-white in their peripheral, and chalky-white in their central, parts. The corneas were available for histologic examination, which revealed infiltration of the stroma with neutral fat and free cholesterol and no signs of an inflammatory process. The author believes that this primary degenerative fatty infiltration of the corneas is comparable to the atheromatous softening of the aorta in old age.

P. C. KRONFELD.

General Diseases

ARTERIAL HYPERTENSION AND THE FUNDUS OCULI. LEOZ ORTIN, Arch. de oftal. hispano-am. **31**: 201 (April) 1931.

After a detailed review of the current views on arterial hypertension, the author considers all noninfectious lesions of the fundus as most probably dependent on arterial hypertension, especially those in connection with albuminuric and diabetic retinites, retinal hemorrhages and transitory amblyopia. Similar processes are to be observed in the choroid.

C. E. FINLAY.

A CASE OF MIKULICZ' DISEASE WITH DISORDERS OF THE EYE. JAGNOV and SEGALLER, Cluj. med. **11**: 420, 1930.

The authors' case showed photophobia, epiphora and multiple infiltrations of both corneas. The disease had existed on both eyes for five months. Palpebral edema, hyperemia and follicles of the conjunctiva had in the beginning suggested the diagnosis of trachoma and indeed caused a mechanical expression of the follicles to be carried out. Then followed a slight transitory improvement of the condition. A general

reaction of the lymph nodes and symmetrical hypertrophy of the preauricular, submaxillary, sublingual and lacrimal glands and of the parotids occurred. Repeated cultures taken from the conjunctival secretion were negative. The Wassermann reaction was negative. Eosinophilia of from 3 to 4 per cent was present in the blood. Hence the diagnosis of trachoma could be ruled out as was that of vernal conjunctivitis and Parinaud's disease. The hypertrophy of the glands, the reaction of the lymph nodes, the eosinophilia and the history (a brother died of lymphogranulomatosis) justified the view that the condition in question was Mikulicz' disease.

N. BLATT.

Glaucoma

GLAUCOMA AND LOW TENSION DISC CUP ENLARGEMENTS. R. PICKARD, Brit. J. Ophth. **15:** 323 (June) 1931.

This paper embodies a plot of the tension against the size of the optic disk cup, with an investigation into the elasticity of the cup, together with an inquiry into the origin of the cases of low tension; it compares the clinical course of the two conditions and discusses the name to be applied to the condition when it occurs with no rise in the intra-ocular tension.

The cubic capacity was calculated on the basis that the transverse diameter of the disk is 1.5 mm., and the vertical, 1.6 mm., and that a depth of —3 diopters equals 1 mm. The results negative the opinion that intra-ocular pressure is an active factor in the enlargement of the cup; if this had been so, the larger cups would have had a pressure near the higher limits of normality; this is not the case.

The estimation of the elasticity of the cup surface was based on a formula suggested by Prof. F. H. Newman that $S\left(\frac{1}{r^1}+\frac{1}{r^2}\right)-t$, in which S = surface tension, r^1 = radius of cup in depth in mm., r^2 = radius of cup in horizontal diameter in mm., and t = intra-ocular tension. The results indicate that there is a great variation in the resistance of the nerve head in otherwise apparently normal nerves, so that more or less equal tension may produce very different results in different eyes.

The evidence is against intra-ocular tension being the cause of the recession of the cup; the lower elasticity in this group suggests an actual decrease in the resistance of the nerve head. The group of cases of small cup found in routine examination of the companion eyes shows that changes have occurred in the optic disk before any alterations of the cup have occurred. Any alteration in the position of a limiting surface fixed at its periphery can only occur as the result of a disturbance of the force acting on either side. In the case of the optic disk cup, the cup enlarges by pressure exerted through the vitreous or by traction by the nerve head tissues: the former has been shown not to be increased; therefore, the enlargement is caused by contraction of the nerve tissues.

The conclusion arrived at is that the primary cause is in the nerve head itself, and is not an effect inflicted on it from without, as is the case in glaucoma.

In glaucoma, the effect of the cup on the nerve fibers, as far as the enlargement is concerned, is limited to changes caused by the longitudi-

nal tension on the fibers, together with the possibility of effects on the nerve fibers produced by the original cause of the recession. In chronic nonintermittent glaucoma it may fairly be stated that blindness is the final inevitable result if the patient lives long enough, a rule with few exceptions. On a priori grounds it may be surmised that this might not be so in the low tension group.

The author suggests that the term "glaucoma" be reserved, as formerly, for cases of increased tension and the sequelae of increased tension. The condition called "cavernous atrophy" by Schnabel fits these cases of recessed cup excellently, whereas it is difficult to see how it can arise in cases of increased tension.

W. ZENTMAYER.

Hygiene, Sociology, Education and History

THE MEDICOLEGAL VALUE OF THE TRAUMATIC SYNDROME OF THE ANTERIOR SEGMENT. H. FRENKEL, Arch. d'opht. **48**: 5 (Jan.) 1931.

In 1916, the author first grouped together under the heading of a traumatic syndrome of the anterior segment a number of conditions that have usually been described separately. In this paper he points out the value of such a general conception from both a theoretical and a practical standpoint. The conception of these cases led him to a different pathologic explanation. He believes that subluxation of the lens is the fundamental factor, and that it occurs much more frequently than is generally recognized. From a practical standpoint, the idea of great frequency of slight subluxation of the lens has led to the description of a new sign in the diagnosis of this condition. It consists in a semicircular fold of the iris situated near the great arterial circle but independent of it. The existence of this sign has been confirmed by Jacquean. A second practical result of the grouping of these cases under a general heading has been a better understanding of traumatic cataracts. He classifies these according to whether they are due to (1) perforating wounds, (2) commotion, (3) indirect contusion or (4) direct contusion. The latter type of cataract is usually partial and rarely becomes complete. A third practical result is the deposition of pigment on the anterior surface of the lens, which may be mistaken for evidence of old iritis. A fourth result is the explanation of traumatic paralytic mydriasis. A fifth has to do with changes in refraction, a sixth with traumatic Argyll Robertson pupils, and a seventh with the loss of central visual acuity, which is out of proportion to lesions of the anterior segment. The eighth and final result has reference to the medicolegal aspect of this symptom grouping. A number of detailed illustrative cases are cited to emphasize its value from the point of view of compensation.

S. B. MARLOW.

INDUSTRIAL REQUIREMENTS IN RELATION TO VISUAL ACUITY. MANACHO, Arch. de oftal. hispano-am. **31**: 135 (March) 1931.

After a detailed study of industrial requirements in connection with visual acuity and its correction, with strabismus and binocular vision and with color perception, the author recommends a visual examination, with a corresponding certification, by an expert, of every apprentice entering

a profession or a trade. The findings should be utilized by both the employer and the employee in the development of the workman's ability. It would be advisable to reserve employments that do not require a special visual acuity for those with visual defects that reduce this. Correction of refractive errors should place the workman within the legal requirements for his work. A revision of the requirements of railroad employees as regards visual acuity and perception of color under different light conditions is necessary. The present extreme visual requirements for aviation and the navy should be maintained in full.

C. E. FINLAY.

Injuries

UNDULATIONS OF THE INTRA-OCULAR PRESSURE IN A NONPERFORATING INJURY OF THE EYE. E. MARX and A. E. QUARTERO, Klin. Monatsbl. f. Augenh. **85:** 758 (Dec.) 1930.

A boy, aged 13, could not see with one of his eyes immediately after it had been struck by a piece of coal. The anterior chamber was filled with blood; the tension was so low that it could not be measured with the tonometer; the cornea and sclera were intact. Retroflexion of the upper portion of the iris could be noticed after absorption of the blood; the pupil was enlarged and of sluggish reaction to light and consensual. Iridodonesis, ruptures in and a detachment of the choroid and hemorrhages in the fundus were observed. The central blood vessels were greatly filled and tortuous. The tension was 130 mm. when taken on the twenty-first day after the injury. The type of tonometer is not mentioned. Following this sudden rise the tension declined to normal within a few days, after which it dropped to an immeasurably low level. This undulation occurred four times in succession during twelve days. The tension of the other eye remained normal. The changes of the intra-ocular tension were due, in the author's opinion, to a repetition of irritations and relaxations of the sympathetic nerves supplying the choroid, the cramp causing a diminution of the tension by contraction of the blood vessels, and the relaxation an increase of the tension owing to a dilatation of the vessels of the choroid.

K. L. STOLL.

Instruments

A NEW PORTABLE CORNEAL MICROSCOPE. C. J. L. BLAIR, Brit. J. Ophth. **15:** 333 (June) 1931.

The instrument for purposes of description may be divided into: the microscope and stand, and the source of illumination. The microscope consists of a small optical unit capable of a magnification of × 10 to × 20, inclusive, mounted on a stand for application to the patient's forehead, and with screw adjustment. The stand itself is capable of being adjusted to meet the discrepancies in the slope of various foreheads. The source of illumination consists of a lamp and its mounting. The lamp is a small, luminous ophthalmoscopic bulb with a single filament in a small cylindric case with a condensing lens that is capable of adjustment. There are two stops, one that gives a sharp line and the other an even disk. The lamp is mounted on a slide traveling on a

quadrant, which combines ease of movement along the quadrant with rigidity in other directions. The lamp has two movements, one a rotation about its own axis. Directions are given for its use.

W. ZENTMAYER.

Lacrimal Apparatus

INTRANASAL DACRYOCYSTOTOMY FOR RELIEF OF LACRIMAL OBSTRUCTION: AN ANALYSIS OF SIXTY-THREE CASES. F. HOLT DIGGLE, Brit. M. J. **1:** 391 (March 7) 1931.

The author has performed seventy-nine West operations on patients who were referred to him from the Manchester Eye Hospital, and the results in sixty-three cases were traced for at least nine months after operation.

As to technic, he has abandoned local anesthesia, as the frontal process of the superior maxilla is frequently too dense to remove comfortably with local anesthesia. Anterior middle turbinectomy is avoided, if possible, and the bone in the nose forming the inner wall of the tear sac is removed by a long, narrow and slightly curved chisel, with which three incisions are made, down to the outer periosteum; the square-shaped piece of bone is then removed. The removal of the inner wall of the tear sac, while it is often the most difficult step in the operation, is done with the aid of Paterson's long tenaculum forceps, which seize this wall, and the protruding portion of the wall of the tear sac is then excised with a very slender, sharp knife. The opening, if necessary, can then be enlarged with a pair of biting forceps. It is important that this opening should be as large as possible and at the most dependent part of the sac. There is practically no after-treatment, as gauze packing is to be avoided.

Of the sixty-three operations, forty-six (73 per cent) were highly successful. Six of the remaining seventeen resulted in partial cure, and in eleven the result was a failure. The author believes it is important to select suitable cases, depending on the condition in the nose; operation should not be done when the tear sac is acutely inflamed, and those cases in which recurrent inflammations have taken place are not so likely to yield successful results.

A. KNAPP.

A CONSIDERATION OF THREE HUNDRED WEST OPERATIONS. ALFREDO ALACINO and MANUEL RODRIGUEZ, Arch. de oftal. hispano-am. **31:** 65 (Feb.) 1931.

The paper deals with the advantages of the West operation as compared with external dacryocystorhinostomy (modified Toti operation), with extirpation of the tear sac and lacrimal gland and with catheterism. The author details points of interest on the preoperative, the operative and postoperative conditions.

C. E. FINLAY.

Lens

CYSTEINE OF THE LENS AND ITS RELATION TO ULTRAVIOLET LIGHT. Y. SHOJI, Arch. d'opht. **48:** 28 (Jan.) 1931.

The author describes the technic employed to determine quantitatively the cysteine present in the lens. He describes experiments showing

the effect of light of different wavelengths on solutions of cysteine. Cysteine is found in the lens in two forms, free and combined. In cases of cataract, the free cysteine disappears first; the combined later. His experiments show that in vitro cysteine is greatly affected by ultraviolet irradiation, and doubtfully so by infra-red and visible rays.

S. B. MARLOW.

CATARACT IN RELATION TO THE ENDOCRINE GLANDS. M. T. FISCHER-GALATZ, Clinique, Paris **25:** 119 (March) 1930.

This edition of *La Clinique* is devoted to endocrinology. A review of cataract and its relation to the subject are discussed. Introductory remarks concern the influence of temperature and changes in concentration of intra-ocular fluids on the lens. Cataract in tetany and in myotonia atrophica and the slit-lamp findings are alluded to. The author feels that from an endocrinologic standpoint the cataract of puberty is the most important type. Ancel and Bouin think that it is due to an insufficiency of the interstitial glands during the evolution of puberty. It is found only on atropine dilatation of the pupil, usually in the nuclear equator or in the cortex disposed in a crown formation, and therefore called coronary by Vogt. It is nearly always bilateral and familial. Horlacher believed that 60.5 per cent of the senile cataracts are formed from the coronary cataract. Harms, Nordman and Bergauer described the disappearance of beginning cataracts after the activation of the glands by vasectomy according to Voronoff's procedure. The methods of Lichtenstern and Doppler, to accomplish vasectomy, are described in brief.

L. L. MAYER.

TWO MINOR ACCIDENTS IN THE EXTRACTION OF CATARACT AND MEANS OF COUNTERACTING THEM. M. MARQUEZ, Arch. de oftal. hispano-am. **31:** 132 (March) 1931.

The accidents in question are the falling of the iris before the knife during the corneal section and an upward subluxation of the cataract on the application of pressure for its expulsion. In connection with the former, the author objects both to the usual procedure of continuing the section and cutting the iris and to the withdrawal of the knife and postponement of the operation; he advises withdrawing the knife with simultaneous enlargements of the puncture and counterpuncture, followed by an introduction of a spatula through both these, with a reintroduction of the knife in front of the spatula and completion of the section with a simultaneous protection of the iris with the spatula. In connection with the second accident, he brings the cataract down with a cystotome and then applies pressure in the usual manner.

C. E. FINLAY.

Lids

XANTHELASMA TREATED WITH THORIUM-X; CASE REPORTS. W. MEERHOFF, Klin Monatsbl. f. Augenh. **85:** 661 (Nov.) 1930.

Operative removal of xanthelasmas does not prevent recurrences, nor is it practical in cases with multiple xanthelasmas. The use of roentgen rays and radium being too risky for the eye, Loehlein experimented with

ray treatments of thorium-x and with an ointment and also an aqueous solution of thorium-x. The solution must be freshly prepared in each instance, because the activity of the alpha rays is at its height twenty hours after preparation, sinks rapidly afterward and is exhausted after two weeks.

Meerhoff reports on six cases treated at Loehlein's clinic by placing small pieces of one layer of gauze, the size of the xanthelasmas, over these formations and by moistening them with the solution from four to five times. The treatment was continued until a cutaneous reaction appeared. The doses administered could be figured out, since the quantity of "es-E" (Emanations Einheiten or emanation units) contained in each cubic centimeter of the solution was known. Most of the xanthelasmas disappeared completely, while some of them remained visible in the form of faint yellow spots. The treatment with thorium in any form, including the aqueous solution, will not prevent recurrences in some cases. It is recommended, however, on account of its simplicity, painlessness and lack of risk. Large doses have not been used up to this time.

K. L. STOLL.

Methods of Examination

CONTRIBUTIONS TO THE INVESTIGATIONS OF THE FUNDUS WITH RED-FREE LIGHT. M. NAKAJIMA, Keijo J. Med. **6**: 437 (Dec.) 1930.

Nakajima describes a simple apparatus for producing red-free light which uses a 250 watt Mazda electric bulb instead of the carbon arc as a source of light. For his filter he uses a 13 per cent copper sulphate solution and a 0.0079 per cent brilliant green water solution, which is cheap and always available, instead of the expensive Zeiss-Vogt glass filters which deteriorate with use.

The theory of red-free ophthalmoscopy and contributions to the literature are briefly summarized.

Nakajima investigated fifty-seven cases of Kakki amblyopia (a manifestation of beriberi), and found, with red-free light, characteristic abnormal superficial retinal reflexes in the papillomacular region, which appeared much earlier than changes seen at the disk (hyperemia, temporal pallor and atrophy) with the usual source of light and which, therefore, permitted treatment to be started before actual nerve fiber destruction had occurred. He classifies the disease as a subacute or chronic variety of axial neuritis.

The author also discusses the findings with red-free light in cases of central retinitis, optic atrophy, Leber's disease and number of other diseases, and describes the appearance of the normal fundus with red-free light.

W. F. DUGGAN.

RADIOGRAPHIC DEMONSTRATIONS IN ORBITOSINUSAL CONDITIONS. C. ANDREESCU, Cluj. med. **11**: 418, 1930.

The article presents radiographs concerning orbitosinusal suppurative lesions (mucocele, pansinusitis with periorbital phlegmon) and tumors of the orbital wall (osteomas, chondromas), all of them made after the new method of Mayer and Moehlmann. Orthoradiographs of

the optic foramen, taken by the method of Rex, are also shown. The author emphasizes the advantages of the axial position, by which procedure the shadows of the bones can be best avoided. N. BLATT.

Ocular Muscles

MINERS' NYSTAGMUS. W. E. COOKE, Brit. M. J. 1:615 (April 11) 1931.

The author concurs in the generally accepted belief that defective illumination is the primary cause of miners' nystagmus. Latent nystagmus, however, may become evident under a variety of conditions—as, for example, during anesthesia, in the course of acute infections, in alcoholism and after trauma. In regard to the last phenomenon, the theoretical explanation has been given that trauma may result in the liberation of protein substances which, on absorption, may exert a toxic effect on the already impaired myoneural apparatus of the eyes.

In an attempt to identify some factor or factors that might be responsible for this transforming of a latent into an evident nystagmus, the author carefully examined a series of fifty-seven persons, all of whom were certificated as incapacitated by the disease.

Such factors as age, family history, color of eyes, diet, consumption of alcohol and tobacco, refractive errors, syphilis, working period, etc., proved negative in the sense that none of them was common to all the cases in the series. The same can be said for the results of the examinations of the central nervous system and of the circulatory, respiratory and renal systems. There was no constant deficiency of the blood serum or of the bleeding time. The only positive finding was in connection with the blood counts. On completion of the series it was found that not one case had a normal Arneth count. In hematologic terms there was a pronounced "shift to the left"; that is, there was a striking predominance of relatively immature forms of polymorphonuclear leukocytes in the circulation. This is held to be the result of a stimulation of the bone marrow, in turn dependent on a toxemia. On surveying the cases with this in mind, it was possible to demonstrate in every case some focus or condition to which a toxemia might be attributed—septic tonsils, pyorrhea, purulent bronchitis, lead poisoning, nephritis and other conditions. The author explains the immunity of the 65 per cent of the miners in whom nystagmus does not develop by attributing it to a personal refractoriness to the effects of toxins. O. P. PERKINS.

A STUDY OF TWO THOUSAND CASES OF MINERS' NYSTAGMUS. J. OHM, Arch. f. Ophth. 125: 245 (Nov.) 1930.

The author is well known for having made innumerable careful studies of miners' nystagmus. After twenty-five years of work on the abnormalities of ocular motility he brings a summary which should be read in detail. He has developed an excellent technic for recording ocular movements, the "lever nystagmography." According to two experts in this field, Fox and Dodge, "Ohm has collected much valuable information by this method, especially concerning the gross changes in

the eye movements under various pathologic states" (Optic Nystagmus: II. Variations in Nystagmographic Records of Eye Movement, *Arch. Neurol. & Psychiat.* **22**: 55 [Jan.] 1929).

P. C. KRONFELD.

Orbit, Eyeball and Accessory Sinuses

FRACTURES OF THE OPTIC CANAL. ROLLET, PAUFIQUE AND LEVY, Arch. d'opht. **47**: 737 (Nov.) 1930.

This study is based on the radiologic observation of ten cases of unilateral optic atrophy of traumatic origin. Although autopsy shows very well the bony lesions, it is difficult to determine the exact mechanism of the ocular disturbance in the patients who survive. In these patients, radiographic study can be of the greatest value. The literature on this subject is reviewed, and the anatomic relationships are pointed out. The radiologic technic is described in great detail, and considerable space is devoted to the interpretation of films. Ten case histories are concisely presented and are discussed from the standpoint of etiologic circumstances, symptomatology and prognosis. In regard to prognosis, the cases fall into two groups: (1) those in which vision is completely abolished from the beginning and in which it never returns, and (2) the cases in which vision is only diminished and there is a chance of improvement, but restitutio ad integrum is exceptional. From the radiologic aspect, the authors believe that a lesion of the optic canal always occurs in these cases. Great care is necessary. In one instance, their confidence in the positiveness of their observations led them to suspect an exaggeration of symptoms, with confirmation after prolonged observation.

They suggest that the pathogenesis of the lesion of the optic nerve is due to the slipping of the bones forming the canal, producing immediate damage to the nerve. They are inclined to discount compression of the nerve by callus, hematoma and detached pieces of bone as causes of injury of the nerve. For this reason they are of the opinion that surgical intervention is unjustified and useless in these cases.

S. B. MARLOW.

Physiology

DARK-ADAPTATION AND PERIPHERAL COLOUR SENSATION OF NORMAL SUBJECTS. R. LYTHGOE, Brit. J. Ophth. **15**: 193 (April) 1931.

After a brief introduction reviewing the observations of Young, Troxler, Purkinje and others on peripheral vision, there follow a historical résumé of the subject and a theoretical consideration of color vision. The author's experiments were designed with a view of finding the fate of day vision (cone or epicritic) during dark-adaptation, and they were a continuation of work on the changes in the critical frequency of flicker during the course of dark-adaptation. Since some of the results were difficult to explain, a parallel investigation on peripheral color sensations was made. The points at issue were (1) the differences in color sensations between the light and dark adapted eye, and (2) the relative behavior of different subjects.

The conclusions drawn from the experiments on the critical frequency of flicker were that the highest perceptible rate of alternation of black and white increases during the course of dark-adaptation for the rods, and decreases for the cones. Evidence is advanced for these conclusions.

The apparatus and nature of the experiments made to compare the sensations evoked by color stimuli in the light and dark adapted eyes are described.

The summary as given by the author is as follows: 1. Experiments were made with a view of determining the influence of the adaptation of the eye on hue discrimination in the central and peripheral parts of the retina. 2. During dark-adaptation color sensations are much desaturated, and hue discrimination is less acute. 3. The possible causes of this diminished acuteness of hue discrimination are discussed. It is due to: (*a*) the suppression of the mechanism of color perception during dark-adaptation, (*b*) swamping of the sensation by an achromatic rod response or (*c*) the presence of pigments within the eye. The results do not point unequivocally to any one of these causes.

W. ZENTMAYER.

Refraction and Accommodation

SPASM OF ACCOMMODATION IN TABES. N. BLATT, Arch. f. Ophth. **125:** 236, 1930.

Blatt describes the findings in a man, 34 years of age, who had tabes dorsalis with typical gastric crises combined with spasms of the accommodation during which the increased curvature of the anterior surface of the lens could be demonstrated by the reduced size of the reflex image on that surface. The dynamic refraction was increased by 6 diopters during the attacks. The patient had Argyll Robertson pupils, which usually contracted to accommodation and relaxed very promptly; during the spasms this reaction was very slow and sluggish in its active and passive phase. The cerebrospinal fluid in this patient indicated a considerable degree of meningeal inflammation. Blatt explains these spasms as a temporary disturbance of the double innervation of the ciliary muscle (stimulation of the oculomotor plus paresis of the sympathetic).

P. C. KRONFELD.

WHAT REGULATIONS ARE EMPLOYED IN PROGRESSIVE JUVENILE MYOPIA? J. MELLER, Wien. klin. Wchnschr. **44:** 21 (Jan. 2) 1931.

A discussion of the proper glasses includes about three fourths of this article. Reference is made to the theory of Javal that convex lenses are indicated, as the accommodation is strengthened by such lenses. The mere fact that concave glasses increase distance vision should be sufficient to satisfy the need for them. Overcorrection with the concave lens is common, but is usually due to incompetence or to mass examinations. No brief is held for the hypothesis that the proper concave lens limits or improves the myopia. There is no truth in the theory that the use of concave lenses will cause decreased visual acuity without glasses. This is simply contrast. The practice of using atropine to paralyze

accommodation over a long period of time has no justification. It does limit and blur close work, which is only apparently improved when the administration of atropine is discontinued, but it does not actually retard the progress of the myopia. The method that attempts to influence distance vision by long practice in viewing test types is a physical impossibility, and no amount of psychic influence is able to change such a condition. Whether the use of proper concave glasses retards the progression is not the question, and this is not a contention. The important fact is that in youth all functions are more amenable to correction, and there is no necessity to allow procrastination until the retinal function is no longer tractable, and changes in external muscles and those of accommodation have taken place. Proper glasses make for correct posture of the head and the entire body. All near work that can be eliminated should be, until a time when myopia is stationary. Myopia does not cause tiredness, as witness the fact that myopic students are usually the more diligent ones. The fact that the nearsighted child sees poorly increases his love for near work, because he finds it more satisfactory than trying to play with his better-sighted friends. It is a common occurrence to find children of near-sighted parents less myopic than either parent, owing to proper care. Meller believes that the type with posterior staphyloma is not amenable to any treatment. The hope that removal of the lens in these cases would be of value has not been fulfilled.

L. L. MAYER.

Retina and Optic Nerve

ANGIOID STREAKS AND THEIR RELATION TO A FORM OF CENTRAL CHOROIDAL DISEASE. R. D. BATTEN, Brit. J. Ophth. **15:** 279 (May) 1931.

From a study of four of his own cases, three of which occurred in the same family, and a consideration of other cases in the literature, Batten gave the following summary: In the first of his cases there were no actual angioid streaks. In the right eye there was a dark mass of pigment similar to the "black spot macula" found in myopes. There was a widespread choroidal atrophy or thinning all around the optic papilla. In the left eye the fundus was similar, but there was no black spot at the macula. Beyond the macula there was a dense, white, crescent-shaped mass, superficial to the retina, ending in feathery tails of retinitis proliferans. A sister and a brother of this patient showed the typical picture of angioid streaks.

This choroidal disease has its onset in middle life, at about 40 years. It is not associated with a syphilitic, tuberculous or septic cause; it is not of inflammatory origin, but is primarily a vascular disease. It is probably inherited, and is often familial. It affects both eyes. The earlier symptoms are a pallor surrounding the optic papilla, fine pigment changes at the macula and central area and angioid streaks, not necessarily with loss of vision. The later symptoms are central macular hemorrhages and exudation associated with retinitis circinata. These in turn tend to absorption, giving rise to extensive choroidal atrophy, with scattered gross pigmentation, and later still to retinitis proliferans. The same choroidal disease occurs without angioid streaks. If the association of angioid streaks with this form of choroidal disease is considered

established, the prognosis in cases showing angioid streaks should be guarded even when there is full vision without symtpoms. The streaks may exist for many years before central hemorrhage occurs.

The paper is illustrated by nineteen black and white and two colored pictures of fundi.

W. ZENTMAYER.

STATUS OF PRESENT KNOWLEDGE AS REGARDS THE TREATMENT OF TABETIC OPTIC NERVE ATROPHY. ANTONIO J. MANES, Arch de oftal. de Buenos Aires **6:** 144 (March) 1931.

After dealing with the existing confusion as to the pathogeny of tabetic optic nerve atrophy, with a slight reference to the symptomatology and the course, the author reviews the treatment followed at different clinics by Igersheimer, Swift Ellis, Bielschowsky, Lauber, Lindner, Fuchs, Elschnig and Fischer with suboccipital injection of arsphenaminized cerebrospinal fluid, combined with insufflation of air, trephining of the sphenoidal sinus, malaria therapy with Lindner's atoxic diet, strychnine and bismuth, with the relatively poor results obtained, concluding that treatment should be mostly prophylactic by intensive treatment of persons freshly infected with syphilis.

C. E. FINLAY.

Trachoma

MARGINAL DIMPLES (FOSSETTES) OF HERBERT AND THE CORNEAL ROSETTES OF TRACHOMA. A. F. MACCALLAN, Rev. internat. du trachome **8:** 53 (April) 1931.

A plea is made that the name of Herbert be continued in association with the limbic changes in trachoma.

The so-called Herbert's peripheral pits, as they are known in Egypt, were first described in 1904 in the *Transactions of the Ophthalmological Society of the United Kingdom* on page 67 of volume 24. MacCallan recalled them to attention in 1913 in the September number of the *Archives d'ophtalmologie* and spoke of their first description by Herbert and later by Meyerhof. They are the result of a cicatrization of the trachomatous follicles of the limbus, are well known and from his experience are found only in trachoma.

L. L. MAYER.

TRACHOMA PROPHYLAXIS IN EGYPT. A. F. MACCALLAN, Rev. internat. du trachome **8:** 54 (April) 1931.

This is a reply to the statement by Morax in the January number of the *Revue International du Trachome,* that "from the point of view of prophylaxis against trachoma, the campaign in Egypt was a failure, and one should profit by this experience in seeking recourse to other methods." MacCallan refers to statements made in his book on trachoma in 1913, shows the tremendous problem and its various means of attack, and finally states that in spite of extreme conditions of poverty and filth, which had existed perhaps five thousand years, nevertheless old residents had remarked that conditions were far superior to those existing thirty years ago.

L. L. MAYER.

PROPERLY SPEAKING, WHAT IS TO BE CALLED TRACHOMA? N. BLATT, Orvosi szemle 3: 343, 1930.

This question has not yet lost anything of its actuality, wherefore it could be made the subject of discussion at any congress. Trachoma as an infectious disease possesses rather an endemic character. In the postwar times as compared with the prewar times there was, in contrast to what has been stated by many authors, no increase in the number of trachomatous patients. The official measures taken against the diffusion of trachoma are, in all countries where they have been enacted, almost identical and tend for the most part to the unitarian conception; i. e., conjunctival folliculosis and trachoma are deemed to be one and the same disease except that folliculosis represents the initial stage of trachoma. The unitarians stress as arguments the fact that both conditions manifest themselves in the form of follicles and show nearly the same location on the conjunctiva. The dualists, on the contrary, base their arguments on a large series of facts; namely, that follicles can occur on the conjunctiva in the most various diseases of the eye; that any tissue, hence the conjunctiva, too, reacts to the most various pathogenic causes with always the same pathologic products; that the follicles represent only a reactive form of the conjunctivae due to any abiologic stimulus; that folliculosis is a benign condition, while trachoma is a malignant one; that folliculosis never leaves scars, while trachoma always does; that folliculosis does not involve the tarsus, while trachoma often involves it, and that folliculosis never entails pannus as does trachoma.

The conception of the unitarians, particularly if adopted officially, may in practice prove injurious; for instance, a patient with folliculosis in a place where there is compulsory treatment may be confined in a hospital for trachoma, where he may really acquire trachoma.

But the therapy, too, of both nosologic forms is different. In regard to the pathology of trachoma the fact is emphasized that pannus may occur in the initial stage. The lacrimal glands and those of Krause and Henle, as well as the meibomian glands, as being the portals of entry for infections of the conjunctiva and especially for diseases of the tarsus, should be given much more attention than has been done hitherto. Hebert's sign, the S-shaped crookedness of the palpebral margin, is due to the involvement of the meibomian glands.

In order to decide if a disease in question is or is not trachoma, the bacteriologic and cytologic examinations (inclusion bodies) are far from being sufficient. The clinical symptoms still play the decisive part, provided, of course, they rest on adequate personal experiences of a physician well acquainted with the question of trachoma. N. BLATT.

Tumors

A CASE OF UNRECOGNIZED JUXTAPAPILLARY SARCOMA OF THE CHOROID. J. CHARAMIS, Arch. d'opht. 47: 833 (Dec.) 1930.

The author describes a case of intra-ocular tumor in which operation had been performed several times for a chronic glaucoma without any signs of new growth until enucleation for absolute painful glaucoma revealed a sarcoma arising from the choroid. The presence of a bilateral glaucoma completely masked the development of the tumor which arose

in the posterior pole from the cribriform layer. The glaucoma had persisted for more than a year and a half. The tumor had not penetrated the sclera or extended into the nerve, although the globe was completely filled. The author discusses the difficulties of diagnosis in cases of this kind. He calls attention to the diminution in elasticity of the sclera on palpation, first described by Terrien, and believes it to be a valuable aid in these cases. Prognosis must be guarded. It is difficult to decide how malignant a tumor may be. The author's patient was alive and in excellent health eight months after enucleation.

S. B. MARLOW.

THE ETIOLOGIC RELATIONSHIP BETWEEN TRAUMA AND SARCOMA OF THE ORBIT. A. TRETTENERO, Ann. di ottal. e clin. ocul. **59:** 272 (March) 1931.

The author's patient was a child of 3 years, who, in a fall, had wounded the right eye and orbit on a sharp object. Traumatic cataract developed, which was removed a month after the accident, with apparently a good result. Two months after the operation, vision had decreased, the tension was raised and right exophthalmos was present. A detachment of the retina was seen which did not transilluminate clearly; therefore, enucleation of the eye alone was performed. Sections showed an intra-ocular tumor which was considered by the author, after study of sections, to be a mixed cell sarcoma. Two months later a new growth was present in the orbit. Evisceration of the orbit was refused, and the child died three months later.

The reported cases of orbital sarcoma following trauma are reviewed. In most cases doubt existed as to the etiologic importance of the trauma. In the author's case the possibility of an etiologic relationship could not be excluded. It is definitely known that trauma may accelerate and aggravate the course of a new growth already present.

S. R. GIFFORD.

TUMORS OF THE CILIARY EPITHELIUM. L. HEINE, Arch. f. Ophth. **125:** 481, 1930.

The author describes a peculiar proliferation of the pigmented layer of the ciliary epithelium which occurs if a retinal detachment extends onto the ciliary body and separates the two epithelial layers. This proliferation presents itself in the sections as a spindle-shaped thickening which resembles a malignant melanoma of the choroid, from which it is separated by the lamina elastica. Secondarily, mesodermal tissue invades this epithelial growth. This growth seems to be the typical reaction of the ciliary epithelium to contact with the subretinal fluid. The author gives a scheme of the reactions of the other parts of the primary retina to the various stimuli.

P. C. KRONFELD.

Sympathetic Ophthalmia

TWO CASES OF CURED SYMPATHETIC OPHTHALMIA. E. PUSCARIU, Cluj. med. **11:** 416, 1930.

A boy, 15 years old, suffered from a perforating injury of his left eye with prolapse of the iris. Therapeutic excision of the prolapsed

portion of the iris, thermocautery and conjunctival autoplasties were employed. After six weeks there appeared a sympathogenie and a sympathetic iridocyclitis, which were treated by atropine, injections of milk, intravenous injections of urotropine, arsphenamine and mercury. After two months he was symptom-free. Vision was: right eye, 1; left eye, 1/6.

A boy, 7 years old, suffered from a severe perforating injury to the left eye. After fifty days there presented a sympathetic ophthalmia of the right eye with uveitis, deposits on Descemet's membrane, synechiae of the iris and a hyotonic painful eyeball. Cure with clearing up of the inflammatory phenomena was secured. The acuteness of vision, however, remained reduced.

N. BLATT.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

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TRANSLATED BY DR. PERCY FRIDENBERG, NEW YORK

(From Summaries by the Readers of the Papers)

TORIC LENSES. DR. BICHELONNE, Paris.

Dr. Bichelonne demonstrated the experimental method used by the Benoist-Berthiot establishment to determine the surface curves of toric lenses. A comparison of toric spherocylinders of a base of 6 diopters with Orthal toric lenses showed that with this method a minimum variation of astigmatism with oblique vision was obtained with the Orthal toric lenses.

AN INTERNATIONAL OPTOMETRIC CHART. DR. DOR, Lyons.

Dr. Dor presented a new international optometric chart at 18 degrees, composed of disks and black squares of equal surface on a basis such that when the disks are superimposed on the squares, the diagonal of the latter exceeds and passes beyond the diameter of the disks by a fraction. This scale, furthermore, has been arranged and graduated on a decimal basis, but presents gradations not found in the usual decimal scales.

A COLOR TEST APPARATUS. DR. POLLACK, Paris.

Dr. Pollack, referring to the theories of Seebeck, presented a color test apparatus which allows one to detect the slightest degree of daltonism. It consists of a box containing an arranging leaf and a collection of small cardboard or wool rectangles suitably colored. Each color forms a scale of at least eight shades of gradually decreasing saturation. A glass plate or suitable screen covers the leaflet and holds the color rectangles in the order in which they are classed by the subject under examination. Under the board or leaflet the bottom of the box is divided into two compartments, one for colors used in a rapid diagnosis, and the second for all others. For the first, coarse test, the colors chosen are those which, in the experience of the authors, are common confusion colors in all cases of color blindness, viz., purple, violet, blue and blue-green. The subject is required to group these colors in monochromatic scales of decreasing saturation. Color blindness is detected more surely and as rapidly, at least, as with the pseudo-isochromatic color charts. By Lumière color photography, permanent records can be made of defective color vision, a study of which may throw new light on the subject of color blindness.

JEHAN YPERMAN. DR. DE METS, Antwerp.

Dr. De Mets commemorated the six hundredth anniversary of the death of the father of Flemish surgery. Until seventy-five years ago, the name of this man had remained unknown. The chance discovery, in England, of a Flemish manuscript which was later brought to Belgium and compared with the archive records revealed the name of Jehan Yperman, born at Ypres about 1280. He studied medicine at Paris, under Lanfranc of Milan. After six years of study he was master of surgery at Ypres, attending at the Hôpital de Belle (destroyed during the siege of Ypres, 1914 to 1918). The manuscript is one of three copies of lectures on surgery and medicine, in which he taught candidates for a degree. In this document one finds, in the chapter on hemostasis, that Yperman knew perfectly, practiced and taught ligation and torsion of arteries according to the classic teaching of the old masters, and that, accordingly, Ambroise Paré is not the inventor, as has been claimed. In the chapter on the eyes, he described external ophthalmia, from the mildest form to those that destroy the eye unless successfully treated. He described "lippitudo" which secretes freely and is contagious, requiring repeated scraping of the flesh which grows within and underneath the substance of the lids. It appears that there were trachoma granulations in Flanders even in those early days. An ungula (pterygium) should be incised. Cataracts may be curable or incurable; the former should be couched with the needle when ripe, but not otherwise. Blindness results when the nerves no longer conduct light to the brain, and this is incurable. Yperman's canon of ophthalmology seems to have been at least as important as that of Guy de Chauliac (1363). This treatise of his is the first medical document written in the popular idiom, and antedates that of Ambroise Paré by two hundred years. If the latter is called the father of French surgery, Yperman certainly deserves the name of father of Flemish surgery.

SODOKU OF THE EYELID. DR. MONTHUS, DR. FAVORY and DR. J. LEVADITI, Paris.

The authors showed lantern slides of a case of sodoku of the lids. Of late, and especially since the World War, infections due to spirilliform organisms have attracted much attention. Among other forms, many cases of sodoku have been reported in France and elsewhere abroad. The case reported was of interest because the point of entry was in the upper lid, and the disease, in spite of arsenical treatment, resulted in death. The pathogenic organism could not be isolated either from the liquid aspirated from the palpebral lesion or from the blood, but the clinical symptoms seem to show beyond a doubt that this was a case of sodoku with primary palpebral involvement.

LYMPHOBLASTOMA OF THE ORBIT. DR. JEAN SEDAN, DR. ASTIER and DR. CAUDIÈRE, Marseilles.

The authors reported a case of lymphoblastoma of the lacrimal sac involving the orbit and spreading and becoming generalized to the entire lymph gland apparatus. A woman, aged 44, presented a lacrimal abscess which was incised; only a small amount of pus was evacuated. By mischance, probes were passed repeatedly (eight times). Thirty days

later, the patient began to see double. A large lobulated mass presented in the region of the sac and the inner aspect of the orbit, and was resected with the lacrimal sac. The tumor was lardaceous. Histologic examination showed that it was composed essentially of lymphoblasts. This lymphoblastoma appeared to be very sensitive to roentgen therapy, which was applied, with medium penetration, for a terrific relapse coming on suddenly three weeks after operative removal of the growth. The tumor literally melted under the rays. Then there appeared, in turn, tumors of the preauricular, submaxillary, cervical, axillary, inguinal and, finally, mediastinal lymph glands. Each of these regions was irradiated successively with complete success and cured, without recurrence in the irradiated regions, but the growth process seemed to flee before the x-rays. Cachexia and death followed the mediastinal involvement. Two biopsies of glands removed for examination confirmed the diagnosis, histologic and clinical, of lymphoblastoma.

LATENT INFECTION OF THE LACRIMAL PASSAGES. DR. E. KALT and DR. MARCEL KALT, Paris.

Serious complications in the form of iridocyclitis following ophthalmic operations are generally attributed to conjunctival infections, and we learned long ago that it is dangerous to operate on a watering eye. Even without evident tearing or conjunctival inflammation, a large number of eyes are menaced by latent lacrimal infection. Fluid injected into the lower canaliculus passes into the nose with difficulty or not at all. Bacteriologic examination of the flora of the conjunctival sac gives uncertain results. The authors reported on the examination of thirty lacrimal sacs, extirpated previous to operation for cataract. They found that the entrance of the canaliculus into the sac was the seat of an infiltration by plastic cells, and later, this was complicated by an invasion of lymphoid nodules, evidence of a serious infection. This proceeded downward without any evident dilatation. The epithelium was destroyed and the surface of the sac resembled a fleshy bud which was later replaced by a cicatricial layer; hence, stenosis and obliteration of the sac. Neither probing nor antiseptic injections can cure this trouble. It is absolutely necessary, for security, to extirpate the sac. The results obtained were most favorable. This descending infection, which begins at the inner conjunctival culdesac, may be called stenosing follicular dacryocystitis.

DIATHERMY IN THE TREATMENT FOR OBLITERATION OF THE LACRIMAL PASSAGES. DR. DEFOUG, Laon.

Basing his method on urologic technic, on account of the analogy of the symptoms requiring treatment, the author passes a high frequency current through the lacrimal probe in the treatment for epiphora. The treatment lasts about ten minutes and is repeated at intervals of from five to seven days. The electrization causes no pain or faradic sensation. The probe plays the part of an active electrode, the subjective reaction allowing one to estimate the temperature of the thermophore. Results are obtained rapidly and prove to be lasting. A single treatment is often sufficient in cases of recent catarrhal tearing. Acute dacryocystitis is favorably affected, while even the most obstinate chronic dacryocystitis

yields to several diathermic catheterizations. This procedure is attractive, easy, effectual and absolutely harmless, and ought to become a routine practice.

LATE COMPLICATION OF DACRYOCYSTORHINOSTOMY. DR. MARIN-AMAT, Madrid, Spain.

In two cases, five days after operation, the patients were suddenly awakened from deep sleep at midnight by a profuse nasal hemorrhage which did not yield to general medication with vasoconstricting drugs and plugging of the anterior and posterior nares. In one case, the hemorrhage gradually subsided in the course of a few days, leaving general anemia which persisted for some time. In the second case, the loss of blood was extraordinary, exsanguinating the patient and necessitating, in a condition of collapse threatening fatal issue, the renewed baring of the field of operation, breaking down of the intervening bone and direct compression of the bleeding artery, which was situated under the middle turbinate and had not been reached by the tampon of gauze introduced through the nostril. In this operation, an upper and a lower horizontal incision and at the same time a vertical one are made in the nasal mucosa in order to get a quadrangular flap. With the lower horizontal incision, some branch of the anterior ethmoidal artery, which supplies this region and follows a vertical course, is probably cut. As the patient's general condition improved and the blood pressure rose, the spontaneous postoperative clot separated or disappeared, allowing the vessel to reopen and bleed freely, as its position under the middle turbinate prevented its being tamponned by the wick of gauze introduced through the nostril.

TUBERCULOSIS OF THE CONJUNCTIVA. DR. CUENOD and DR. ROGER NATAF, Tunis.

The authors presented a study of the differential diagnosis between trachoma and tuberculosis of the conjunctiva based on biomicroscopy. In one case they were able to follow the progress of the lesions from their beginning. The tubercles appear, at first, in the form of small, depressed necrotic areas. These depressions are each surrounded, little by little, with a sort of bolster of projecting follicles so as to form a budding mass with a depressed, umbilicated center. At the periphery of these true tuberculomas, the vascularization, which is very slight to begin with, does not later get to the point of forming true papillae, in mosaic. The lesions then undergo regressive metamorphosis, and this resorption is marked by the disappearance of the details just described. Once formed, these tuberculous growths, it is to be noted, are much larger than trachoma granulations.

PARINAUD'S CONJUNCTIVITIS. DR. SZYMANSKI, Vilna.

A case of Parinaud's conjunctivitis was reported in a child of 8 years with edema of the lids, unilateral granuloverrucose vegetations and swollen lymph gland pockets on the same side. Histologic examination showed plasma cells. The absence of necrotic foci and giant cells, and

recovery in five weeks without scar formation disprove the German hypothesis of a tuberculous origin and confirm the theory of Morax.

FILTRATION OBTAINED BY SCLERAL TREPHINING. DR. ROCHON-DUVIGNEAUD, Paris.

The author considered true and false filtering cicatrices produced by scleral trephining. Filtration may persist indefinitely with hypotony, or obliteration may take place so that tension rises above normal. In the former case, the conjunctiva is generally raised up over a large area, extending more or less beyond the scleral opening. In the other case, the conjunctiva may be raised, but its margin is adherent to that of the scleral opening so that the bleb does not extend beyond the latter, and does not allow filtration of the aqueous into the subconjunctival tissue. If, in this case, one again dissects up the conjunctival bleb flap the scleral opening is found wide open, the aqueous escapes, and tension goes down. If the flap is then sutured into place, filtration persists in a certain number of cases, and a new fistulization of the eye can be obtained without a second trephining operation.

TUBERCULOUS SCLEROKERATITIS TREATED WITH METHYLIC ANTIGEN OF NEGRE AND BOQUET. DR. COLRAT, Lyons.

The author reported two cases of tuberculous sclerokeratitis in women, aged 28 and 32, respectively, giving the typical symptoms and clinical course. Bilateral, multiple inflammatory foci were noted in the sclera close to the limbus, with interstitial infiltration of the corresponding areas of the cornea; repeated attacks occurred with the usual accompanying iritis. In one case, there were intracorneal tubercles. After recovery, there was a persistent deep vascularization of the cornea throughout its entire extent. Both patients had a tuberculous heritage and personal history. Treatment with methylic antigen caused no reaction, arrested the lesions and brought about a rapid clearing of the cornea. It would seem to be the method of choice in tuberculous sclerokeratitis.

DEEP ULCER OF THE CORNEA. DR. VALIÈRE-VIALEIX, Limoges.

The author called attention, on the basis of a large number of cases seen within the last few years, to the marked difference in clinical course and in prognosis between superficial, serpiginous ulcers of the cornea and deep infectious abscess. The former are easily reached by surgical means, particularly cauterization, and generally yield to the traditional treatment. The deep ulcer, of which the author reports sixteen cases, spreads in the interstitial tissues and often leads to complete invasion of the cornea and adherent leukoma. Surgical intervention seems to stir up the purulent process and to do more harm than good. Cauterization, and particularly paracentesis, should be avoided. After treating the lacrimal passages and disinfecting the conjunctival sac, one should perform an extended blepharorrhaphy of the external canthus. If this does not stop the purulent process, it at least has the advantage of maintaining perfect occlusion, protecting the globe in case of sudden per-

foration which may lead to extrusion of the lens and vitreous, as happened in two of the author's cases.

PROGRESSIVE ULCER OF THE CORNEA. DR. R. RUBBRECHT, Bruges, Belgium.

Dr. Rubbrecht described a new form of treatment for progressive ulcers of the cornea. Spontaneous perforation and vascularization favor the cure of this condition. With the operation described, the author aimed to imitate this combination of factors. The ulcer is, first, carefully curetted, and then sectioned from in front backward, the incision extending into sound tissue beyond the ulcer, on either side. The corneal lamellae are cut on the bias, and the line of the incision never passes at any point through the center of the cornea. In this way, prolapse of iris is prevented, and coaptation of the lips of the wound is assured. The ulcer is then covered with a conjunctival flap with a double pedicle. This supplies protection and the vascularization necessary for the repair of tissue and for resorption. Even the most virulent ulcers generally heal under this treatment, the purulent process is checked, the opacity does not extend beyond the limits of the corneal ulcer, and there is no opalescent loss of transparence around it. Later on, there is considerable clearing. The operation is indicated in all forms of progressive ulceration.

THE RÔLE OF HEREDITY IN OPHTHALMOLOGY. DR. MARNIX VAN DUYSE, Ghent, Belgium.

Heredity in General and Its Pathogenesis; Laws of Hybridization.—Heredity, in its biologic sense, is the transmission to the offspring of a certain number of the characteristics of the parent. With this transmission there is associated resemblance, noted repeatedly and recorded in the adage: Like father, like son. But, on closer observation, this resemblance is not complete and absolute; between children of the same union there are often considerable differences. For a long time, this transmission from one generation to another and the differences appearing in the young of the same family were attributed to chance, for lack of a better explanation. In 1865, a monk, Gregor Mendel, a learned botanist and mathematician, found in studying the variations in character in hybrid plants that these manifestations of heredity, far from being due to a caprice of nature, were subject to fixed rules, which could be formulated as laws. Mendel's work was the starting point of a new science of heredity. This science has not been slow to advance, with the progress of the natural sciences, particularly biology, the introduction of experimental methods in botany and zoology, the development of microscopic anatomy and discoveries in cytology and microchemistry. The basic fact that all living beings, vegetable, animal and human, show similar vital phenomena and are made of cellular elements of the same nature, allows one to draw analogies between the manifestations of heredity noted in the field of botany and zoology, and those seen in man. Mendel's experiments did not, at first, attract much attention. His laws had, as it were, to be discovered again and brought to the notice of the scientific world, by the work of Correns, Tchermak and de Vries.

Mendel's laws of hybridization include monohybridism, disjunction of characteristics, purity of gametes, the law of proportional numbers and intermediary hybrids. The simplest example of monohybridism, due to the English botanist, Correns, is the crossing of two plants of identical, pure race and distinct coloration, pink, and white, respectively. The plants issuing from this crossing all have pale pink flowers and constitute the first hybrid generation. A second hybrid generation, obtained by crossing these hybrid flowers among themselves, results in three varieties, one with pink flowers, like the maternal ancestor, one with white flowers, like the paternal ancestor, and one with pale pink flowers, like the first hybrid generation, in the proportion of 1, 1 and 2. Each plant results from the union of two reproductive cells or gametes, male and female, which have the faculty of reproducing the specific racial characteristics as well as certain special features of family or stock. In the example just cited, the parental plants are of a pure race, i. e., the issue of gametes having identical aptitudes, homogametes or homozygotes (Bateson); members of the first hybrid generation (F_1) are heterogametes or heterozygotes and show without exception an intermediary coloration. Disjunction of characteristics occurs in the second hybrid generation, showing that the gametes are pure and that each of them carries only a single aptitude. In each generation a certain number (one-half) of plants having both kinds of gametes will produce mixed progeny, while the pure one-fourth will produce pure red, and one-fourth pure white, i. e., the colors of the original parental generation, without a trace of the previous crossing. This proportion is noted in successive generations. The crossings described produce no persistent variation. The descendents take over the characteristics of the parents, and the intermediary aspect never appears in plants of pure race. In some cases it is impossible to distinguish hybrids by coloration, as the parental hue may persist, as a dominant, while the maternal hue is recessive, but the law of proportional representation persists unchanged. Similarly, of two or more inherited characteristics, one may be dominant and overlie the others, so that the inheritance is not apparent and may become so only on further experimental crossing. The distinctive characteristics of each of the races originally crossed are distributed independent of each other in the reproductive cells (independent segregation of characters). This is of importance for the transmission of hereditary characteristics in man. As the number of independent hereditary characteristics increases, the make-up of the generation F_2 becomes more and more complicated. If the two crossed races have, for example, ten different characteristics, F_2 will show 2^{10}, or 1,024 individuals differing in appearance. It is well established that the germ cells, male and female, are the basis of assimilation as well as of heredity and that the nucleus plays the all-important rôle in the formation of daughter cells. The chromatin of the nucleus divides by mitosis or karyokinesis in such a way that the nuclear substance is divided with absolute equality, and the number of chromosomes formed by the fragmentation of the original nucleus is constant for a given animal or vegetable species. The chromosome composition of a gamete is called simple or haploid; that of the fecundated cell is diploid; its nucleus resulting from the union of two gametes contains twice the number of chromosomes. Mendelian disjunction is based on the reductive division of the chromatin of the

fecundated female cell, the heterozygote (hz), and the redistribution of this chromatin among the gametes of the heterozygote. The chromosome composition of the haploids reproduces all possible combinations between the maternal and the paternal chromosomes. The influence of consanguinity is not deleterious, ipso facto. It is nil in the case of families without stigmas and of restricted influence in the case of pathologic characters which are dominant. Its importance is noted, however, in recessive characteristics, in which case the pathologic manifestations are noted often in the children of blood relations. When they appear for the first time, one must assume that they have previously existed in the ascendants and are able to remain latent for several generations. Familial diseases act like recessive characteristics. Developmental defects producing malformations noted at birth are truly hereditary if they depend on pathologic changes inherent in the germ cell, male or female. Other malformations, usually superficial, such as coloboma of the lids or lacrimal passages, may be due to defects of the amnion. Other influences may act in utero, such as trauma or decidual hemorrhages, and fetal disease or inflammation may play a rôle.

The heredity of ocular diseases is discussed as follows:

Familial Diseases with Continuous Heredity.—Hesperanopia: In this form of heredity, only affected subjects can transmit the disease. Truc-Nettleship-Capion's disease, or essential hesperanopia, has been followed through so many generations from the original patient, Nougaret, that it can be definitely stated that normal subjects never transmit the disease. It must be differentiated from acute forms, such as those due to avitaminosis, hepatic disease and other general conditions, and it is definitely hereditary, congenital and essential, the subjects being absolutely healthy, intelligent, vigorous and of average longevity. It is also regional, being limited to a rather small part of southern France, in the Cevennes. There are no visible ocular lesions. Ophthalmoscopic examination gives negative results. Errors of refraction are negligible. Central vision is always good. Night blindness is marked. There is direct hereditary transmission as a dominant characteristic, either sex being affected. There is another form of hesperanopia which is regularly accompanied by myopia, and which is transmitteed as a sex-linked recessive characteristic, as well as a form seen with high myopia and transmitted as a simple, not sex-linked recessive (Vogt).

Cataracts: These are transmitted as dominants. Congenital forms involve only the zone of the embryonic nucleus, and the designation congenital cataract should be applied only to total or to zonular cataracts. Zonular Cataract: This form may remain stationary for a long time, the opacity being located in the layers surrounding the embryonic nucleus like a shell or sheath, while the center remains clear. It generally appears at birth, rarely later. Horner attributed zonular cataract to rickets; Peters, to tetany; others accuse disturbances of nutrition or of endocrine balance. Baur felt that a combination of these causes may be in play. Other forms of congenital opacity are stationary and so of minor clinical importance. Those which were formerly called anterior and posterior polar cataracts trace their origin back to remains of the fetal pupillary membrane or the hyaloid artery and have the characteristics of congenital anomalies. They are located in Vogt's cortical layers. Knies noted, in one and the same family, an anterior capsular opacity alternating with zonular cataract, and Vogt reported each of these mani-

festations in two successive generations. They seem to be related, as is fusiform cataract, to other forms of congenital cataract. Biomicroscopy has shown opacities limited to the capsule in apparently normal lenses in members of families in whom only pyramidal cataract had been observed. Before the introduction of the slit-lamp these persons would have been considered normal. Ziegler and Griscom described a case of posterior opacity which was transmitted as a dominant characteristic. The affected nucleus proved the congenital nature of the case.

Intracrystalline Opacities: The three conditions described by Vogt are probably different localizations of a single disease: (*a*) an embryonic anterior axial cataract which is above all things stationary, does not affect vision and is not a cataract in the true sense of a progressive opacity of the lens; it is situated in the neighborhood of the anterior Y, and is composed of small white deposits not over 0.1 mm. in diameter; (*b*) a central punctate dustlike cataract made up of extremely fine, minute points of opacity, occupying the embryonic nucleus (Vogt's layer 3) in front of the (clear) interval; (*c*) opacity of the sutures taking on color under intense illumination and situated in the anterior or posterior Y, or in both. This last form is related to fusiform cataract. Stationary opacities of the lens in adults have a close resemblance to the congenital variety, being, like the latter, small, clearly defined, scattered and of variable form and progressing very slowly if at all. The one point of difference is that they never develop in the fetal nucleus as do true congenital opacities, but only in the adult nucleus or in the cortical layers. Still, both forms are probably due to the same etiologic factor, acting in one case before, and in the other after, birth. The first and most common form of senile opacity consists of a fine white dust or powder, each grain of which is so minute that it is impossible to assign a definite shape to it, scattered in the cortical layer and in the adult nucleus, and invisible to the naked eye as well as to the ophthalmoscope. This opacity is stationary and does not affect sight. A second form appears on examination with the slit-lamp in the form of dotted opacities of various forms, dimensions and arrangement, and has been described under different names; one, opacity of the sutures, is of definitely congenital origin.

Floriform Cataract (Koby): Numerous opacities sketched out congenitally continue to appear during the formation of the adult nucleus and of the cortical layers; they are made up of rounded and annular elements, giving to each of them a polycyclic, floriform appearance. They are clear to transmitted light (diaphanous to transparence), bluish-white on oblique illumination and yellow under reflected light. Most of them are grouped about the anterior and posterior sutures of the fetal nucleus, whose Y form they sometimes assume, but similar opacities are found in the deeper cortical layers, indicating that they have developed since birth. This form has been observed in three successive generations.

Cataracta Dilacerata (Vogt): This is composed of peculiarly marked opacities, with tattered margins, pierced with holes, the opacities being extremely minute and giving blue or green reflexes with focal illumination; they are located both in the deep layers of the adult nucleus and in the superficial layers of the fetal nucleus and do not progress. This form is intermediate between congenital opacities and the stationary forms in adults.

Cataracta Punctata: Opaque dots more or less numerous in the anterior layers of the lens are irregularly scattered mostly in the deep cortex, and do not progress. Vogt's cataract in crown form is made up of club-shaped opacities arranged in a sort of wreath about the equator of the lens with the thick end toward the center, combined with round opacities of a bluish or yellowish hue (cataracta punctata cerulea). They are situated exactly at the border of the adult nucleus and the cortical layers; they are generally claviform, with the large extremity turned toward the center of the lens. Vogt considered this form a familial disease. Halbersma found in a family of seventy-five, a punctate cataract alternating with zonular cataract in forty-nine.

Senile Cataract: The older clinical nomenclature spoke of cataracts as cortical, perinuclear or nuclear according to the localization of the opacities, at the beginning, in this or that level of the lens. The first two forms are the more frequent and common (92 per cent of a series of 166 cases). Biomicroscopy has shown that there are only two forms of progressive, senile, lenticular opacification. One starts in the non-sclerosed cortical or perinuclear layers, producing the various forms of cortical cataract; the other appears in the center of the lens, in sclerosed nuclear layers, and progresses toward the periphery, producing nuclear cataract.

Cortical Cataracts: Their common characteristic seems to be the development on a basis of vacuoles, clear spaces (fissures), separation of sutures and lamellar dissociation, which have been attributed to hydration of the lens due to absorption of fluid. They start in the cortical layers or the most superficial of the adult nucleus, anterior or posterior, most commonly in the region of the equator; they are never blue, but of a grayish hue, thickly massed, dense and rather coarse. As they develop, the lens swells somewhat (intumescent stage) and their progress is rather rapid. The lens may shrink later (detumescence) to its original size. The elasticity of the capsule then plays a rôle and prevents the formation of folds, preserving the original aspect, but by this time opacification is complete. Cortical cataract may start as (1) cuneiform or wedge-shaped cataract, with sector-like opacities in the deep layers of the cortex near the equator, ending in a point directed toward the center of the lens. This is the commonest form. The opacities are dense, white or mother-of-pearl (P. H. F.) and situated in the posterior as well as the anterior layers. Straddling the equator, their appearance is rather different from that of Vogt's claviform cataract. (2) Dust-like cataract in concentric layers begins with an opacification of the middle layers or, according to Vogt, in the deep cortical layers and at the equator of the adult nucleus (perinuclear variety). (3) Posterior cupuliform cataract, Vogt's posterior saucer-like cataract, occurs in the deep posterior layers of the cortex (4) and following a layer or stratum located beneath the posterior capsule, often difficult to distinguish from a pathologic, secondary cataract or a traumatic form. The forms described relate only to the initial stages of a cataract, and their distinctive signs are merged in others and are effaced as the cataract develops and other opacities appear. The last stage is complete opacification of all superficial layers up to the crystalloid, and the cataract is ripe. The genealogical tree of Grote shows that the parents of an affected person may be free. Baur claimed that one is not entitled to infer from this

a recessive transmission, as the parents, had they lived, might have developed cataract at a later age.

Nuclear Cataract: Located in already sclerosed lens layers, the diffuse opacities with extremely fine elements are quite different from the coarser deposits noted in cortical cataract. They are independent of vacuoles, clear spaces and lamellar dissociation; they appear to have nothing to do with absorption of fluid, and do not pass through a phase of intumescence. At first, there is a rapid increase in optical density of the nucleus, and later, progressive opacification. This form progresses very slowly. It may even be stationary, and the generally quite aged patients have very poor vision. The factor of heredity is not very apparent in senile cataract, and the study is difficult as a single observer has no chance to follow up more than one or two generations, at best. The members of a family may die before reaching the age at which they might have been affected. The few data gathered speak for a dominant inheritance. Clausen noted that it is usually the same type of cataract which appears in different members of the same family. There is a form of hereditary cataract the progress of which in young subjects is like that of progressive senile cataract. A peculiar occurrence, anticipation, has been noted in the appearance of senile cataract at a more and more early age, in successive generations. Norrie reported a striking instance followed through five generations, in the last of which the cataract appeared at birth. Baur was skeptical as to the theory of anticipation. He called attention to the fact that blind persons and those with very poor sight are less apt to marry than normal persons. If, carrying cataract determinants, they mate, they do so at an age when the pathologic determinant has not yet become manifest, and so the couple really constitute a sort of selection for the late appearance of the disease. Among their descendants, this selection no longer exists, and the disease shows itself at an earlier age and gives the impression of an anticipation, without any biologic basis. No case like Norrie's has been carefully observed since more has been known about the modes and mechanism of heredity. If these cases were more frequent, Baur said, he would try to explain them thus: A determinant of cataract which produces the disease when in the heterogamete (htg) stage may produce a cataract at an early age, when it becomes homogamete (hmg). Disease of a recessive type may be transmitted in a latent state through several generations and finally become manifest by homozygoty (hmz) in several members of the family. A similar manifestation may result in organisms with a dominant heredity; an even more striking anticipation would be the result. As to the causes of noncongenital cataract, senility cannot be the only cause, as so many old people, aged 80 and more, remain free. Leaving out such exogenous factors as radiant heat (bottle-blowers, locomotive firemen, etc.), one may consider internal secretions (von Hippel, Siegrist) or a theory of heredity based on the determinants being below par. The latter view is supported by the fact that members of the same family are always affected at a definite age (homochronous heredity), as in other hereditary manifestations due to an influence of the genotypes, and it is generally the same type of cataract that appears with a rather extensive variability and not infrequently associated with other developmental defects of which it may be the cause or the result.

Congenital Ectopy of the Lens: The simple form is transmitted in most cases as a dominant characteristic (Franceschetti) and to as many as four generations. An irregular dominance is at times noted, as free subjects may transmit the disease, so that the recessive mode is apparent in analogy with complicated ectopia. This is often associated with other congenital anomalies. Ectopy of the lens and pupil appearing in many individuals of a generation seems to show transmission by recession. In two cases (Franceschetti) the parents were blood relations. Continuous heredity seems to be exceptional. Correlation with colobomas, changes in the lens, particularly in the posterior capsule, which may progress, myopia, megalocornea and miosis (very slight dilation of the pupil under atropine) have been noted.

Family Degeneration of the Cornea (Fleischer): The commonest form, Fuchs' nodular keratitis, is characterized by the appearance of small, round, irregular, grayish opacities in the parenchyma of the cornea. The smallest dots are seen at the periphery where they are arranged in an irregular circle. The epithelium is raised over these deposits, and vision is affected by the irregularity of the corneal surface. There is a light uniform clouding between the opacities. The disease has been noted principally in males, starting in youth and persisting throughout life. From time to time there may be slight inflammatory attacks with some increase in opacity. In grill-like keratitis the opacities are situated in the thickness of the cornea with fine striations running between them. The epithelium remains intact (Dimmer, Haab). Fleischer considered both these forms as hereditary, presenting, as they do, many points of similarity. There are many intermediary, atypical forms. Fehr had a case in which the opacities were more numerous and made up of minute white confluent points situated at the center, not at the periphery, of the cornea. Franceschetti was of the opinion that these diseases are transmitted as dominants. They are frequently seen in a number of successive generations and, as noted in other hereditary diseases, it is generally the same clinical type of degenerescence that appears in affected members of the same family. Another noteworthy fact is that the subjects become affected at a definitely predetermined age, always the same; i. e., the disease is homochronous. The family tree plotted by Treitscheller involves four families with a common ancestor. The disease did not reappear until the fourth generation. This unexplained feature has also been noted in other familial degenerations, such as Friedreich's ataxia and dystrophia myotonica. The appearance, almost simultaneously, in the descendants of four families suggests an inferiority of the genotype, comparable to developmental defects and abiotrophies. Clausen noted the correlation of this disease with other congenital anomalies (psychopathic constitution, mental disturbances, peaked skull, harelip).

Glaucoma: Clausen thought that both forms of adult glaucoma—the inflammatory and simple—particularly the latter—are hereditary in certain families. The disease has been noted repeatedly in two and three, rarely in more than four, generations. This is explained by its comparative infrequency. The transmission is probably as a dominant as is the case with simple glaucoma, of which Kuemmer noted the direct transmission in two generations. When a certain type of glaucoma

appears in a family, it is always the same type which is transmitted to succeeding generations. Exceptionally, both forms appear in the same family (Nettleship). Anticipation was noted in the inflammatory form of this disease by von Graefe. Children with parents and grandparents who had become affected at 60 and at 50 years of age, respectively, developed glaucoma at 30. If primary glaucoma were always transmitted in exactly the same way, one could conclude that it was a definite morbid entity, *sui generis,* and different from other forms of glaucoma. The family trees so far plotted do not, however, allow one to draw definite conclusions, as they have not been drawn up with the degree of accuracy required by the present day science of heredity. Clausen thought glaucoma a hereditary disease in which the determinant alone is transmitted. Other factors, hereditary or acquired, intervene to favor the action of the determinant, a theoretical, complicated heredity.

Juvenile Glaucoma: The earlier glaucoma appears, the more surely one may infer a hereditary influence. Lohlein tried to make a distinction between juvenile glaucoma, hereditary adult glaucoma and hydrophthalmos, but both forms of hereditary glaucoma, juvenile and adult, have been noted in successive generations of a single family, so that one must admit that they develop on the same genotypic basis. For juvenile glaucoma, direct heredity has been found in 20 per cent of the cases; also collateral heredity, and a combination of both forms of transmission. It is not certain whether it is dominant or recessive. Hydrophthalmos may come on as late as 20, and be mistaken for juvenile glaucoma or even, in anticipation, for adult glaucoma as early as the fifth or tenth year. Clausen considered all three forms of glaucoma as different degrees of a single malformation. In hydrophthalmos it is developed ad maximum; the elimination of aqueous is hampered from the beginning. In juvenile glaucoma it is less pronounced and at first causes no functional disturbance. For this, a further morbid factor is requisite, such as an endocrine disturbance coming on at a certain period, either at the beginning or at the end of puberty. Finally, in adult glaucoma the malformation is at a minimum. The functional disturbance, extremely reduced, does not become manifest until senile involution comes to play its part. If Clausen's explanation of the appearance of the three forms of hypertension at different and definite age periods is correct, the mode of transmission of all forms of hereditary glaucoma should likewise be identical. Association of juvenile glaucoma with myopia has been noted in 50 per cent of the cases. From the biogenetic point of view it is doubtful whether, as some authors claim, the myopia in these cases is due to the glaucoma. Between myopia and the most diverse developmental defects, such as hemeralopia and opaque nerve fibers, there is a widely extended relationship for which it is at least a question whether the same causal factor is involved. Lohlein found hereditary juvenile glaucoma accompanied in half the cases by developmental defects, such as coloboma of the iris, lens and optic nerve sheaths, persistent hyaloid artery, etc. Glaucoma cannot very well be considered as a result of these malformations. It is, rather, a question of a hereditary correlation of developmental defects, analogous to those noted with hydrophthalmos and occasionally accompanying glaucoma in the adult.

Diseases of the Sclera (blue sclerotics, fragilitas ossium, otosclerosis [van der Hoeve's syndrome]): The first report is that of Eddowes

(1900). In 1896, Spurway reported four generations of a family presenting the first two of these three clinical manifestations. Peters (1908) reported blue sclerotics in four generations. Van der Hoeve (1917) added, as a characteristic symptom, diminished hearing, which forms the triad that bears his name. The blue coloration is the most constant feature and is due less to an actual thinning than to transparence of the sclera (Vogt). The latter is not actually colored blue but the phenomenon of diffusion, like that which causes the iris to appear blue, produces the effect by the deeper-seated uveal pigment shining through the tissue of the stroma. The bones fracture under the slightest traumatic provocation. This may be due to a progressive osteoporosis or to some disturbance of organic calcium production, with other bone defects, frontal or occipital bosses, dislocations, anomalous movements of the articulations and sometimes dwarfism. The poor hearing is due to otosclerosis, caused by morbid changes in the structure of the petrous portion and calcareous degeneration of the labyrinth, combined partly with ankylosis of the stapes. Delayed development of the teeth and their brittleness are also associated with a disturbance of calcium production. (This may also explain the comparative transparency of the sclera [P. H. F.]). Other ocular congenital anomalies are associated, such as arcus senilis, keratoconus, glioma, choroidal atrophy or zonular cataract, or there is syndactylia, mongolian idiocy, hemophilia, cardiac lesions, cleft palate, etc. The clinical picture represents a hereditary mesodermic modification (Eddowes, Peters) causing the osseous and sclerotic anomalies. Dyscrinism has also been claimed, the disturbance of calcium assimilation being ascribed to lowered function of the suprarenal capsules.

Diseases of the Lacrimal Apparatus: Heredity is of importance in their genesis. Nieden noted familial manifestations in 90 per cent of the cases. Dacryocystitis neonatorum, due to an occlusion of the nasolacrimal canal, has been noted (Groenouw, Peters) in several infants of the same generation, more rarely in the ascendancy of affected subjects. Schnyder saw disease of the lacrimal sac in adults of two families. In the first, the disease had been transmitted for three generations, once by an immune subject; in the other, several members of a single generation showed the condition, which had appeared first between the ages of 20 and 40. Congenital absence of the four puncta lacrimalia and of the lacrimal canals was noted by Emmert in a boy whose brother had a lacrimal fistula. It was due to stenosis of the nasolacrimal canal. The grandfather had suffered with epiphora. Congenital atresia of the canaliculus was noted by Groenouw and Peters in infants of the same generation.

Familial Diseases with Discontinuous Heredity (Mendelian Recessive Type).—These diseases may be transmitted by immune subjects, and reappear after having spared one or more generations. A typical example is offered by albinism. Albinism: This condition appears in man and animals and may be partial or total. In the former case the pupil is not black but pinkish, due to the fact that light diffusing through the sclera and irregularly reflected within the eye comes out through the pupil (and faintly through the nonpigmented iris [P. H. F.]) to the eye of the observer. Ophthalmoscopic examination reveals the margin

of the lens, and at times the ciliary processes and the zonule. The fundus is extremely light; all the choroidal vessels are seen. There is generally marked photophobia, vision is much reduced and the condition is frequently accompanied by nystagmus and strabismus. The lack of pigment involves not only the uveal tract but the lashes, eyebrows and all the hairy structures. The etiology is still obscure, but the rôle of heredity is undeniable, as proved by data of observation in man, and by the fact that in rabbits, for instance, one can breed a stock absolutely deprived of pigment. The disease is rare in Europe (1 to 20,000 or 30,000 [Pearson]). Poor sight is due not only to lack of pigment but to loss of the macular reflex (Ichikawa) and of the luteal yellow (Vogt). Histologic examination has borne out these clinical observations (Elschnig, Fritsch, Fuchs, Velhagen). Data of more than 700 genealogies collected, mostly from English and American sources, by Seyfarth prove beyond doubt that albinism is transmitted as a simple recessive characteristic according to mendelian laws. The appearance of the disease is favored by (1) the mating of heterozygote individuals (DR $\times$ DR) and appears the more frequently (from 25 to 35 per cent) the greater the proportion of heterozygotes is to normal subjects; (2) by consanguinity, submitting the offspring to a "convergent" cumulative heredity and increasing their chances of being affected by a malady latent in the family; (3) by the mating of homozygote subjects (RR $\times$ RR). Tertsch's genealogy shows: (*a*) indirect heredity (mating of cousins), the father and mother being latent carriers (DR); among their offspring were one albino and two normal children; (*b*) direct heredity (mating of uncle and niece); of six children, four were albinos. The mother was albinotic (hmzRR); the father, a latent carrier (htzDR). Here, consanguinity favored the meeting of recessive characteristics. If the parents had not both been tainted, the recessive characteristic could not well have become manifest. The influence of consanguinity on the appearance of a pathologic character is minimal when this character is widespread, but, on the contrary, marked if the character is unusual. According to Davenport, 33 per cent of albinos are born of consanguineous matings. The danger of near approach of pathologic determinants is particularly great in case the father and mother are closely related. In animals, as in man, crossing of albinos produces, exclusively, albinos (mating of hmzsRR $\times$ RR). There are various forms of incomplete albinism which are not transmitted from one generation to another, like the total form, as recessive: (*a*) monocular albinism with absence of the macula (Makulalosigkeit), often with astigmatism and sometimes with nystagmus; transmission as a dominant; (*b*) the fundus only is albinotic, the macula rudimentary with, occasionally, high myopia; sex-linked transmission; (*c*) macular aplasia without pigmentary defect in the fundus (Koyanagi also noted nystagmus in two children of blood relations); it is probably recessive; in this form the hair and skin are very slightly pigmented; it is correlated with albinism.

Retinal Pigmentary Degenerations: The predisposition to this gradually progressive disease is inborn, but it generally becomes manifest later, at the age of 12 or more. Night blindness (hesperanopia) is the first symptom. Changes in the visual field, with an annular scotoma progressing toward the periphery and toward the center, come on later, and central vision becomes affected in proportion to the contraction of

the field. It may be good for a long time, but is generally completely lost toward the fifties. Before this, the patient, although able to tell time on a watch, is unable to find his way about. The characteristic appearance and peripheral location of the pigment, the association with choroidal vascular sclerosis and intervascular depigmentation are well known. Very rarely, macular involvement is noted. The waxy yellow color of the secondarily atrophic nerve head is typical. The disk margin is slightly indistinct. Hyaline degeneration of the walls of the arteries, with thickening, causes a decided narrowing and diminution of caliber. In the periphery most arteries are threadlike; others have entirely disappeared. There is atrophy of the nervous elements of the retina, the fiber layer alone remaining, with hyperplasia of the supporting connective tissue. The atrophy of the ganglion cells leads to degeneration of the axis cylinders and ascending atrophy of the optic nerve. The choroid shows vascular sclerosis, pigment proliferation, and concretions (hyaloid, colloid) in the lamina vitrea. Gowers was the first to formulate the theory of a special hereditary predisposition by which a tissue or an organ may undergo degeneration which comes on just when its full development has been reached. The pathologic manifestations are: 1. There is proliferation of the neuroglia like that which occurs in many forms of nerve abiotrophy, but differing from that observed in sclerosis. 2. Pigmentation of the retina occurs, preceded by atrophy of the rods and cones and their nuclei (external granular layer), which produces gaps in the external limiting membrane through which epithelial cells loaded with pigment and pigment granules migrate by virtue of the physiologic property of all pigment to wander toward the light. 3. The faintly colored spots of the retina and the hyaline nodules on the disk are due to degeneration of cells of the pigment epithelium, which in the immediate neighborhood of the optic papilla are cemented to the choroid and the neuroglia. In typical cases the process starts in the periphery and gradually extends toward the central regions and the macula which have developed later. An infantile form has been seen with simultaneous involvement of the periphery and macula leading rapidly to complete blindness. As the macular changes cannot be detected with ordinary illumination, this form is often overlooked. The pathologic pigmentation which comes on later has been taken for the first morbid manifestation and the disease incorrectly referred to the juvenile or classic form studied by Nettleship, Usher and J. Bell. This form is transmitted as a recessive, as shown by its appearance in many children of a single couple (Donders, von Graefe) and the frequency with which consanguinity has been noted in the ascendancy. Its appearance in two successive generations does not necessarily prove dominant transmission, as has been supposed, as this may be due to the mating of a homozygote with a heterozygote. Sex-linked heredity was noted by Nettleship and and by Usher. There is, finally, a senile form, noted by Nettleship in a person between 50 and 60; this comes on more slowly and with less marked symptoms. The simultaneous or alternating appearance of retinitis pigmentosa, retinitis pigmentosa sine pigmento, and retinitis albescens in children of the same family suggests filiation of these diseases (Clausen).

Oguchi's Disease: This is characterized by night blindness with a peculiar grayish-white appearance of the fundus, which may disappear after several hours of clouding, the ophthalmoscopic picture becoming

normal in this event (Mizeo phenomenon). Histologic examination (Lagrange, H.: *Bull. Soc. d'opht. de Paris,* November, 1929) shows changes in the cells of the pigment epithelium. A few examples of this disease (cases of sclerosis), which is transmitted as a simple recessive (Kawakami), have been observed in Europe.

Amaurotic Family Idiocy (Tay-Sachs' Disease): This condition is generally noted from three to six months after birth. A juvenile form appears later, between the ages of 4 and 16 or more (Spielmeyer-Vogt). The cherry red spot at the macula and simple atrophy of the disk are well known. Clinically, there are associated disturbances of motility with spastic or flaccid paralyses and digestive disturbances. The course of the disease is characterized by rapidly progressive amaurosis, brought to an early end by dementia and spasms, with death from cachexia at the end of the second year. The disease is not frequent, about one hundred cases having been observed, the great majority, but by no means all, among Eastern Jews. The retina is thickened by edematous infiltration of the external molecular layer and by hyperplasia of the ganglion cells. The macula is edematous and shows an increase in the ganglion cell layer. At the center these are absent, and the transparent retina allows the red of the choroid to be seen as the characteristic cherry-colored spot. Recent studies have shown that the retinal lesions are identical with those found in the central nervous system (degeneration of large ganglion cells, followed by atrophy of nerve fibers) and involve not only the macula but the periphery as well. Later the disk becomes atrophic and white. The chiasm and optic tracts are also involved. Sachs accepts the theory of agenesis, or lack of development. Schaffer supposes hypertrophy of the hyaloplasm (Quellungsphase) as a basis for hereditary diseases of a degenerative nature, followed by loss of lecithinoid corpuscles (Faellungsphase). The pathologic process affects only tissues of ectodermal origin (masses of gray matter and cerebral layers of the retina). Marinesco feels that there is a decomposition of the ferments necessary for nutritive exchange of the nerve cell. Parhon and Golstein suggest the possibility of a poison with a selective action on the nervous system. Gordon suggested an endocrine disturbance. This abiotrophy (Edinger) is analogous to retinitis pigmentosa with which it is occasionally associated. The disease is distinctly familial. Two or three children of one mating may be affected, and the appearance in four different branches of a family (Goldfehder) shows the hereditary nature beyond any doubt. Consanguinity has been noted but rarely (four cases in 64 [Apert]).

Juvenile Amaurotic Idiocy (Vogt-Spielmeyer): This form usually comes on later (from 4 to 16 years of age), progresses more slowly, but with the same clinical symptoms, and does not lead to death for a number of years. The lesions of the fundus are somewhat different, as there is secondary pigmentation, which at times involves the macula and at others, the periphery. The histologic changes in the ganglion cells are the same in both clinical forms, but in the juvenile form the neuroepithelium of the macular region (cones and inner granular layer) is markedly changed.

Hydrophthalmos: This congenital anomaly, leading to conditions like those in glaucoma, is due to a malformation of the filtration angle, absence or backward displacement of Schlemm's canal, abnormal per-

sistent fetal ligamentum pectinatum or defect of differentiation of the tissues of the iris angle. Association with other developmental defects, such as irideremia, coloboma of the iris, lens and ciliary body and lenticonus, is common, and this indicates the factor of heredity which is important in so many cases of defective development. The disease may not become manifest until some time after birth, but is without doubt present potentially, and is more frequent in males (63 per cent, according to Seefelder). In 1909, Vogt observed hydrophthalmic rabbits. Crossing one of them with a normal animal resulted in an immune first generation, but mating of animals of the first generation showed hydrophthalmos in one fourth of the offspring. Mating of two affected rabbits caused the anomaly to appear in all the offspring a few weeks after birth, associated, curiously enough, with ribbon-shaped keratitis. Consanguinity, favoring the meeting of determinants, had brought out the recessive characteristics, and so explains the whole litter being affected. Hereditary transmission in man probably takes place in the same way, as a simple recessive.

Directory of Ophthalmologic Societies *

FOREIGN

British Medical Association, Section on Ophthalmology

President: Mr. E. E. Maddox, Glenartney, Poole Road, Bournemouth, England.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Leipzig. Time: June, 1932.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. F. Barnhill, Miami Beach, Fla.
President-Elect: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June, 1932.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Walter F. Hoffmann, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Sioux Valley Eye and Ear Academy

President: Dr. Claude T. Uren, City National Bank Bldg., Omaha.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Fontanelle Hotel, Omaha. Time: November, 1931.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

Southwestern Michigan Triological Society

President: Dr. C. Ward Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the Month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Douglas F. Wood, 74 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.

Montana Academy of Oto-Ophthalmology

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: W. L. Diven, National Bank Bldg., Bismarck.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. M. Hendershott, 193, 11th St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

Utah Ophthalmological Society

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. L. Smith, 1005 Medical Arts Bldg., Salt Lake City.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer. Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Charles W. Buvinger, S. Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society. Section on Ophthalmology

Chairman: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. E. Burner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadephia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Ivor Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831, 5th Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Forth Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert F. Wolters, 130 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassiday, 1620 Medical Arts Bldg., Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Martin H. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

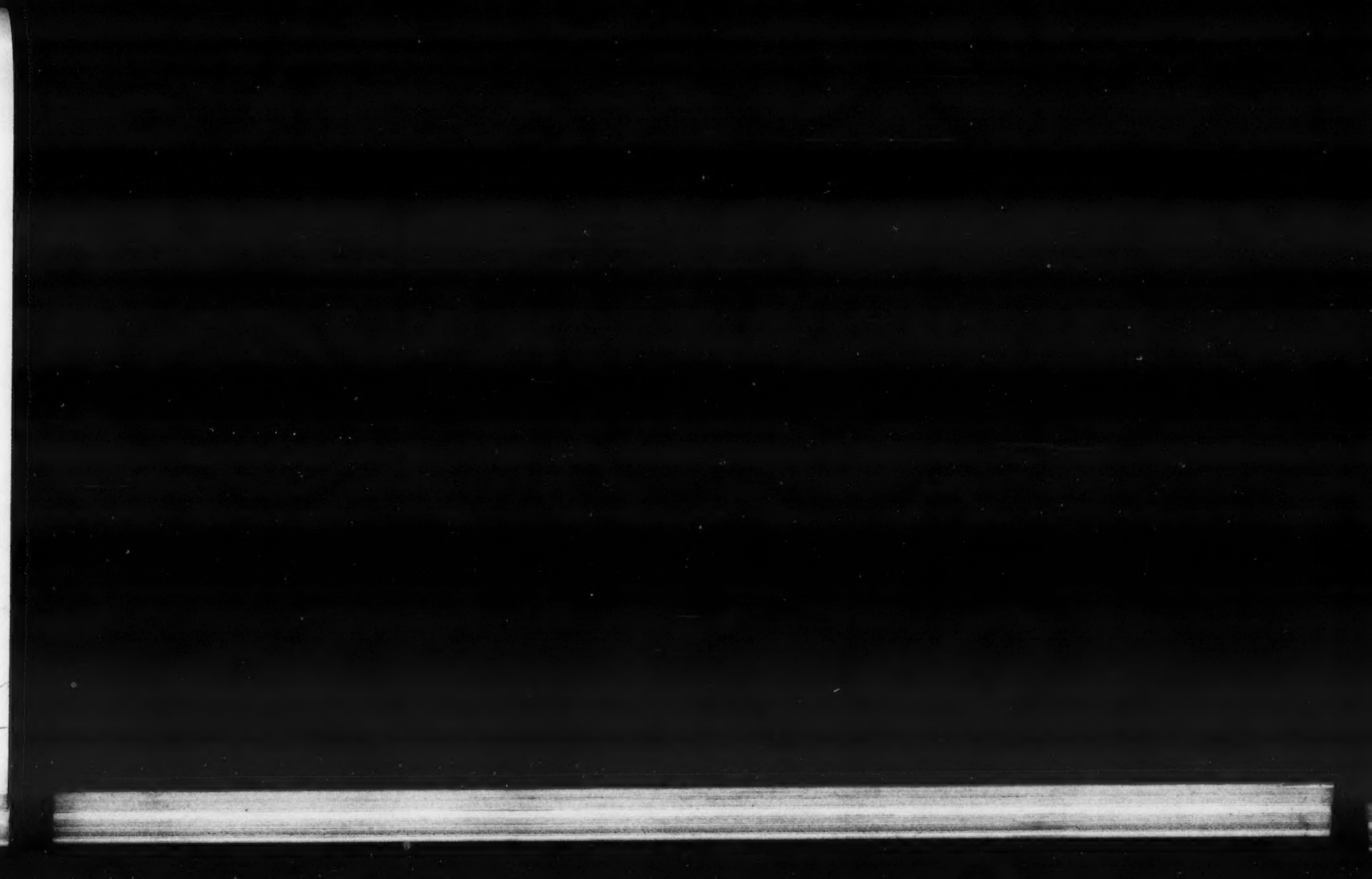
Chairman: Dr. W. F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. Claude L. La Rue, Highland Clinic, Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse.
Place: University Club. Time: Second Friday of each month except June, July and August.